The New England JOURNAL of MEDICINE

FORMERLY THE BOSTON MEDICAL AND SURGICAL JOURNAL

Official Organ of The Massachusetts Medical Society
The New Hampshire Medical Society — The Vermont State Medical Society
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OWNED AND PUBLISHED BY THE MASSACHUSETTS MEDICAL SOCIETY

Volume 208 Number 13 | Domestic $6 per Annum, 25c per Copy Canada $7.04, Foreign $8.52 | MARCH 30, 1933 | Published Weekly in Boston at 8 The Fenway

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VOLUME 208 MARCH 30, 1933 NUMBER 13

NEW ENGLAND SURGICAL SOCIETY

LOW PERICARDIOTOMY FOR ACUTE SUPPURATIVE PERICARDITIS*

Report of Two Cases and Twenty-four New Cases From the Literature

BY P. E. TRUESDALE, M.D.†

ON December 23, 1909 I. H., aged 24, was admitted to St. Anne's Hospital with empyema following an attack of pneumonia. His pulse was rapid. There was a perceptible degree of cyanosis and dyspnea if the patient turned from side to side. The area of cardiac dullness was considerably increased on both sides of the sternum. Heart sounds were scarcely audible. The diagnosis was pericarditis with effusion.

I opened the pericardium through an incision over the fourth and fifth costal cartilages. A massive exudate of pus and fibrin welled out through the incision. The cut edges of the pericardium were sutured to the margins of the wound. With release of intrapericardial pressure, the pulse, respirations, and color of the patient improved temporarily. In spite of repeated irrigations, however, the pericardial sac was never emptied. The heart could be seen partially submerged in pus. The culture showed pneumococcus.

The patient died January 9, 1910, sixteen days after operation.

This experience made a deep impression upon me as a death which might have been avoided by proper drainage. As the literature shows, cases of acute suppurative pericarditis are rare. I did not see another until March 17, 1932 when B. F., an American schoolboy of eight, was admitted to our hospital for surgical treatment of acute suppurative pericarditis. His temperature was 104°, pulse 130, and respirations 44. He had been ill for ten days with a lobar pneumonia on the left side.

This boy showed the immediate effects of a severe pulmonary infection with continuation of acute symptoms. His face was flushed, respirations rapid and labored. There was a perceptible degree of cyanosis. Examination of the chest revealed dullness over the entire lower two-thirds of the left side both anteriorly and posteriorly. Breath sounds were decreased and the heart sounds were weak and distant. The leucocyte count was 35,000. A roentgenogram showed a dense shadow of the pericardium enlarged laterally to about 14 cm. (fig. 1). Pus was obtained by paracentesis.

A blood transfusion was given on March 17. The choice of incision for approaching the pericardial cavity created a lively discussion among members of the hospital staff. Only two approaches were considered, the vertical parasternal, and the horizontal lateral, below the left nipple extending to the axillary line. The lateral approach was discarded.

FIGURE 1.

With the treatment of Case I still fresh in mind, I advocated a low horizontal incision to reach the dependent portion of the pericardial sac. The prevailing sentiment, however, was against opening the pleura and thus augmenting a suppurative pericarditis by an empyema, especially in the presence of a resolving pneumonia. Therefore, in spite of my prejudice against the vertical parasternal incision, I was easily persuaded to abandon my long-treasured theory through fear of making a bad situation worse. With the patient anesthetized by avertin, I made an incision at the conventional site, resecting the fourth and fifth costal cartilages (fig. 2). When the pericardium was opened, approximately 2,500 cc. of pus escaped under great tension. The exudate was thick and contained many large clumps of fibrin. A rubber dam drain was inserted to a point below the apex of the heart.

In spite of the large quantity of pus evacuated, the temperature remained almost stationary at 102°. The pulse rate was 120 but improved in quality. The respirations were 38-40. The pericardial sac was irrigated daily with a one per cent solution of boric acid. Eight days after operation the temperature was 104°. The aperture in the chest wall contracted and appeared inadequate. At each examination this opening was found occluded or nearly so

*Read before the annual meeting of the New England Surgical Society, at Hartford, Conn., October 1, 1932.

†Truesdale—Surgeon, Fall River, Massachusetts. For record and address of author see "This Week's Issue," page 708.

with clumps of fibrin. This interference continued as the temperature pursued an erratic course. To facilitate drainage we used a self-retaining abdominal retractor, introducing the blades under primary anesthesia. The enlargement of the opening thus obtained made irrigation more effective in dislodging fibrin and in reducing the purulent contents of the pericardium. The temperature, pulse, and respiration, however, showed no improvement. The indwelling retractor caused considerable pain and had to be removed.

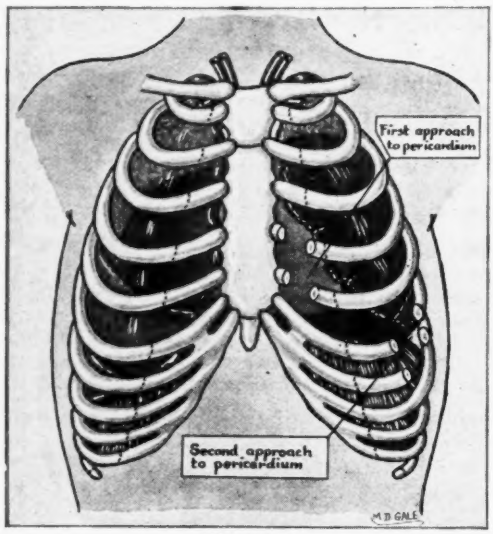

FIGURE 2.

The temperature was still fluctuating widely between 100° and 104° and the patient's condition became alarming. On April 9, the twentieth day after operation, the temperature reached 105° with pulse 140 and respiration 50. The outlook was less hopeful than at any time.

With the heart obviously laboring in a reservoir of pus, I still felt that the mechanism of drainage was faulty and that a change of procedure was warranted even at this eleventh hour. Therefore, with the parents' consent, we prepared to drain the pericardium at its dependent point. The patient was given another blood transfusion after which a horizontal incision was made in the seventh interspace, beginning in the nipple line and extending outward to the posterior axillary line. About two inches of the seventh and eighth ribs were resected (fig. 2).

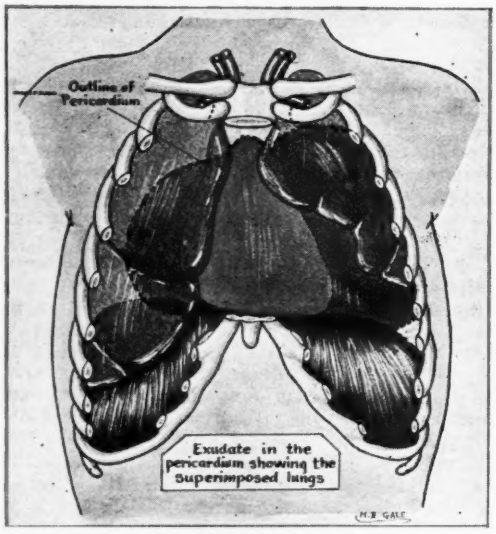

FIGURE 3.

Upon opening the pleura, we met with a surprise. Instead of entering a free pleural cavity, the pericardium was seen with its lower surface resting on the dome of the diaphragm and its outer border extending to the left virtually in contact with and adherent to the parietal pleura. The base of the left lung spread over the pericardium and was adherent to it (fig. 3). The adhesions thus formed completely excluded the general pleural cavity. The edge of the lung had to be dissected upwards in order to afford room for a generous opening of the pericardium. When the sac was incised, a large amount of pus and clumps of fibrin escaped. It was obvious that no more than 50 per cent of the contents of the pericardium had drained through the upper incision at any time. Through-and-through irrigations were employed for a few days only. The discharge rapidly diminished, showing further contraction of the pericardium. The temperature approached normal and the general improvement of the patient went on to complete recovery. Fig. 4 shows the degree of improvement four months after the patient left the hospital.

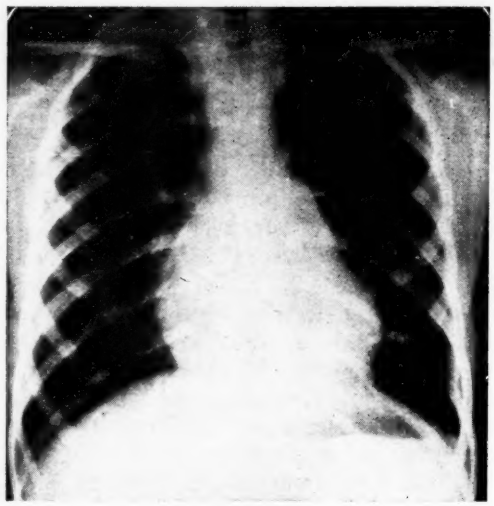

FIGURE 4.

DISCUSSION

In the entire series of 152 operative cases of acute suppurative pericarditis reported thus far, the pericardium, almost without exception, was approached through parasternal incisions. It is clear, therefore, that surgeons have adopted the same principles of drainage in dealing with this condition as those employed in cases of deep-seated collections of pus within the abdomen. Hence it is pertinent to consider the difference in the mechanical forces governing the escape of fluid in the two cavities. By virtue of a varying degree of positive pressure within the abdomen and a negative pressure within the thorax, drainage from a high point is favored in the former and impeded in the latter. The artificially expanded lung, if not adherent, will force free fluid out of the pleural cavity through an opening in the thoracic wall, but has very little influence in forcing a pericardial exudate through an opening at the level of the fourth and fifth costal cartilages. The downward pressure of the normally expanding lung is of great assistance in evacuating contents of the peri-

cardium only when the opening is adequate and made at the most dependent point in the pericardial sac. Hence, Whittemore[1] and Harloe[2] have employed a suction apparatus to overcome this obstacle in draining the pericardium at a higher level.

Is it probable that we have been misled into a belief that posture, irrigations, or suction are good substitutes for gravity in obtaining prompt and thorough drainage of the pericardium? Danger of contaminating a healthy pleura, no doubt, is the deterring factor. In my second case, I deliberately chose to infect the pleura in order to empty the pericardium of its purulent exudate. In both cases clumps of fibrin were so large that they became wedged in the upper incision and completely obstructed drainage. Under such circumstances to be effective, suction would have to be induced through a large tube, in fact so large that its continued use might result in damage to the heart. In Peterson's cases[3], posture accomplished good drainage when the patient could be made to lie procumbent. One can understand this to be true when the opening in the pericardium is large and when the exudate is all fluid. The heart lies anteriorly, however, and when the patient is in a prone position it may act as a valve to close the aperture in the thoracic wall. Irrigation seldom does more than remove the upper level of the residue in the pericardial cavity. Irrigation and suction, therefore, are of doubtful expediency in obtaining adequate drainage of the pericardium, especially when fibrin is contained in quantity.

Allingham[4], believing the low point in the distended pericardium to be behind the sternum, used an approach at the costoxiphoid angle along the lower edge of the costal cartilage. T. M. Thomas[5] in 1929 adopted this method and was obliged to make a second incision over the eighth rib posteriorly. His patient recovered. Thus Thomas and the writer independently solved the problem of drainage in the same way.

Though adhesions excluding the general pleural cavity as found in Case II may be exceptional, it is probable that in the absence of this barrier little added risk is encountered in draining the pericardium through the most dependent point in the left pleural cage. The danger from inadequate drainage through a parasternal incision greatly exceeds that incurred by a low lateral incision even though it might produce empyema. It is doubtful if the first incision in Case II was a factor in promoting the patient's recovery. It was palliative, not curative. Without the second, death was a certainty.

The eritical nature of acute suppurative pericarditis as revealed in the literature gives rise to serious contemplation. Galen recognized the disease in animals and suggested that it might occur also in human beings. The mockery of its appearance in the wake of a resolving pneumonia is a challenge to the instrumentalities of medicine. In Osler's words[6], "No serious disease is so frequently overlooked." Poynton[7] was able to recognize it only six times in 100 autopsies in children. At the Boston City Hospital the correct diagnosis was made in only 17 per cent of 153 cases. There are only 152 operative cases in the literature. Riolan[9] in 1649 first suggested treatment by trephining the sternum. Romero[10] did the first pericardiotomy in 1819. Hilsman of Kiel[11] operated upon the first purulent effusion in 1844. The operative mortality has been reduced from 65 per cent in the first 52 cases reported by Porter[12] and Roberts[13] to 42 per cent for the entire series of 152 cases.

Osler stated that recovery is due not so much to the approach employed or the technique of drainage as to the time at which the operation is done and the condition of the patient following the primary disease to which pericarditis is secondary. Hedblom[14] shows the importance of the primary process in influencing the prognosis. Only five per cent of 51 patients suffering from pericarditis following rheumatism died, whereas when the condition was associated with other etiologic conditions such as empyema, pneumonia, or septicemia, 80 per cent of 40 patients died. Such figures should prompt caution in claiming a recovery from any particular method of drainage. However, the principles of drainage in this affliction may be vital because acute suppurative pericarditis is a condition in which, if principles are disregarded, the chances of failure are greatly multiplied. In the first collection of 52 cases recorded by as many surgeons[15], approach to the pericardium was made through an incision at the left of the sternum in 50. Thirty-two died, a mortality of 64 per cent.

In the next 22 cases published by Elsworth Eliot in 1909[16] the anterior incision was made in 18. In one instance the pericardium was opened through an empyema cavity with recovery. In this series the mortality was 50 per cent.

An additional 13 cases were reported by Rhodes in 1915[17] including two from his own service. The mortality was 46 per cent. In this series there appeared a tendency to approach the level of the diaphragm in the length of the anterior incision.

In 1921 Pool[18] added 17 cases to the literature. The mortality was 47 per cent. The anterior incision in some form was used in all of the operations. Both of Pool's own cases were complicated by empyema. To approach the pericardial exudate in the first, he removed the sixth costal cartilage. This patient died. In the second he made the hockey-stick incision used by Rhodes. This patient recovered.

In Williamson's series[19] of 17 cases, four are

excluded because the treatment was aspiration only. In two of the 13 cases several operations were necessary to drain abscesses lying posteriorly outside the pericardium between the base of the lungs and the dome of the diaphragm. Both patients recovered.

Peterson[20] reports three personal cases, two successful, in which to secure better drainage, the patients were turned on their faces for one-half hour every three or four hours. This method was used effectively by Dickenson in 1889. Peterson believes that when wide exposure and good postural drainage can be established, the use of any sort of tube is unnecessary, if not actually harmful.

Winslow and Shipley[21] reported a series of ten cases, making a total of 126 reported up to September, 1927. There were six cures and four deaths. Shipley reports seven cases, Holland, Lynn, and Stone, one each. In the first four the transsternal route was used. This method of trephining the sternum, successful in two of the four cases, was discarded for avenues giving greater access to the pericardium.

A recent series of six operative cases was reported by White in April, 1932[22]. It is interesting to note that one of his cases was converted into an empyema and the patient recovered.

We have collected 24 additional cases of acute suppurative pericarditis reported during the last five years. Fifteen recovered. The mortality in this series was 38 per cent. There was revealed a tendency to seek a lower level of the pericardium for drainage although the usual anterior incision was employed in 23 of the 24 cases. Shipley[23] since the publishing of his series in 1927 has operated upon four other patients with acute suppurative pericarditis following pneumonia. In two of the four cases the combined transsternal and chondroxiphoid approach was employed. A boy, 17 years old, made an excellent recovery after a stormy, early convalescence with no subsequent cardiac disability. A man of 50 survived operation only 12 hours. In addition to the pyopericardium there was a walled-off empyema. The pericardial sac contained a large quantity of thick pus and there were numerous adhesions between the two layers of the pericardium.

A young woman, 19 years of age, made a slow recovery with subsequent dyspnea on exertion. In this case Shipley used the chondroxiphoid approach and drained the distended pericardium with two Dakin tubes placed behind the heart with the ends sutured within the incision. After a few months, the dyspnea disappeared. Now after two years the patient is apparently well and has completed her training as nurse in an emergency hospital.

The fourth patient was a colored woman, 20 years of age. The effusion was moderately large and the pus thick and creamy. The patient died the day following operation. As in the third case, the simple chondroxiphoid approach was used. Thus, six of eleven patients operated upon by Shipley recovered, the most advantageous method of operation being the combined transsternal and chondroxiphoid route.

In Louck's[24] first case the anterior incision failed to promote recovery and he resorted to posterior drainage of the pericardium through a thoracotomy wound which had been made for empyema. In his second case the incision was made through the posterior axillary line. He perforated the lung in approaching the pericardium. The patient died. In commenting on his experience the author states that posterior drainage has certain natural advantages. It is the method of choice only in cases in which because of an existing pleuritis or empyema there is reasonable evidence that the main pleural cavity has been sealed off from the operative field.

SUMMARY

1. Acute suppurative pericarditis commonly is a secondary infection attacking a patient when his vital capacity has been reduced by the primary infection.
2. It is insidious, concealed, progressive, and without intervention, almost invariably fatal.
3. The infection is seldom recognized clinically. Diagnosis is not difficult if the examiner knows what to look for.
4. The mortality among those treated by operation varies from 38 to 65 per cent.
5. Next to early diagnosis and immediate intervention, adequate drainage is the essential factor in recovery.
6. The extreme lateral and most dependent area of the pericardium is the point of election for drainage.
7. Incision of the pericardium at its most dependent point not less than six centimeters long through a correspondingly large incision in the chest wall is advocated.

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LIST OF CASES OF ACUTE SUPPURATIVE PERICARDITIS (SINCE 1927)

	Author	Sex	Age	Incision	End-result
1.	Ramond and Weill-Spire[1]	M	23	Xiphoid-sternal.	Recovered
2.	Albrecht[2]	M	23	Costo-xiphoid incision.	Recovered
3.	Albrecht	M	19	Recurrent acute pericarditis. Bullet in lung. Multiple rib resections.	Recovered
4.	von Eiselsberg[3]	M	?	4th rib resected.	Recovered
5.	von Eiselsberg	M	19	5th cartilage resected.	Recovered
6.	A. J. and A. S. Welch[4]	F	30	After miscarriage; transsternal route.	Died
7.	Harrington[5]	M	27	Rt. empyema, l. pleural effusion. 7th rib resected in post. axillary line for empyema. Ant. pericardiotomy, 6th cartilage resected.	Recovered
8.	Harloe[6]	F	6	5th left interspace. Trocar and cannula (suction). Dakinization. Hemolytic streptococcus.	Died
9.	Harloe	M	31	Same operation as above.	Recovered
10.	Thomas[7]	F	4 yrs. 10 mos.	Allingham's incision. Second incision, 8th rib resected in post. axillary line.	Recovered
11.	Arntzenius[8]	F	11	Trap-door incision. Staphylococcus aureus.	Died 4 wks. after op.
12.	Loucks[9]	M	19	4th rib and cartilage resected. Thoracotomy for empyema (8th rib). Pericardium drained further posteriorly. Sinuses from osteomyelitic rib; bronchial fistula.	Recovered in 8 mos.
13.	Loucks	M	30	Rib resected in midaxillary and postaxillary line. Lung perforated post. Pericardium drained. T. B. psoas abscess; multiple abscesses.	Died
14.	Brenizer[10]	F	20	Puncture first. Resection of 5th and 6th cartilages. Abscess at puncture site.	Died
15.	Grimault[11]	M	30	Resected 5th and 6th cartilages.	Recovered
16.	Dujol[12]	F	?	Staphylococcic pericarditis, postabortal infection.	?
17.	Garot[13]	Child	4	?	?
18.	Cairo[14]	?	?	Transdiaphragmatic sub-xiphoid.	Recovered
19.	Miller[15]	M	8	5th and 6th ribs resected.	Recovered
20.	McDonald[16]	M	8	To left of sternum, intercostal.	Recovered
21.	Shipley[17]	M	17	Transsternal and transchondral.	Recovered
22.	Shipley	M	50	Transsternal and transchondral; empyema.	Died
23.	Shipley	F	19	Chondroxiphoid approach.	Recovered
24.	Shipley	F	20	Chondroxiphoid approach.	Died

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CASES OF PURULENT PERICARDITIS IN THE LITERATURE

	Author	Sex	Age	Incision	End-result
	1-52 Compiled by Porter and Roberts				
1.	Hilsmann, 1844	M	25	4th interspace near S.	Recovery
2.	Langenbeck, 1850	M		At site of 5th rib (bullet).	Recovery
3.	Rosenstein, 1881	M	10	4th interspace near S.	Recovery
4.	West, 1882	M	16	5th interspace near S.	Recovery
5.	West, 1883	M	14	Opened in chest wound.	Died
6.	Scott, 1883	M	6	5th interspace near apex beat.	Recovery
7.	Savory, 1883	M	9	5th interspace in ant. ax. line.	Died
8.	Partzensky, 1883	M	23	4th interspace.	Died
9.	Gussenbauer, 1884	M	13	5th interspace in ant. ax. line.	Recovery
10.	Newman, 1885	M	32	4th right interspace.	Died
11.	Mikhailov, 1885	F	35	4th interspace near S.	Died
12.	Dickenson, 1887	M	10	5th interspace near S.	Recovery
13.	Underhill, 1887	F	6	5th l. interspace near S.	Died
14.	Parker, 1888	F	9	4th interspace, and 4th costal rib resection.	Died
15.	Halsted-Osler, 1890	M	36	4th interspace mid. bet. nipple and sternum.	Died
16.	Delorme and Mignon, 1890	M	19	4th interspace just outside internal mam. vessels.	Died
17.	Davidson, 1890	M	6	5th interspace.	Died
18.	Davidson, 1890	M	6¾	4th interspace 1 in. left edge of sternum.	Recovery
19.	Teale, 1890	F	11	4th interspace 1 in. left edge of sternum.	Died
20.	Deaver, 1890	M	21	5th interspace 3 in. from midline.	Died
21.	Körte, 1891	F	7	5th rib resected, 5 cm.	Died
22.	Klefberg, 1892	M	19	4th interspace.	Died
23.	Edwards, 1892	F	6	¼" within and above portion of apex beat.	Died
24.	Sievers, 1892	F	22	3rd interspace, 2 cm. to left of sternum.	Died
25.	Jacobson, 1892	F	14	5th interspace, rt. side just outside of sternum.	Died
26.	Gabsziewiez, 1892	M	22	5th cartilage, resected.	Recovery
27.	Allen, 1892	M	11	6th rib resected below and inside l. nipple.	Died
28.	Stoker, 1892	M	20	4th interspace mid. bet. nipple and sternum.	Died
29.	Eiselsberg, 1893	M	17	4th rib (cartilage) resected.	Recovery
30.	Robinson, 1893	M	16	6th rib resected.	Recovery
31.	Pepper, 1894	M	19	5th interspace.	Died
32.	Bohm, 1894	M	33	3rd interspace.	Recovery
33.	Porter, 1895	M	26	5th interspace at 1½" left of sternum.	Recovery
34.	Delorme and Mignon, 1895	M	22	4th interspace.	Died
35.	Marsh, 1896	M	14	4th interspace below nipple.	Died
36.	Björkman, 1895	F	12	5th and 6th ribs resected.	Recovery
37.	O'Carroll, 1896			4th interspace 1" int. to nipple line.	Died
38.	Garber, 1897	F	21	5th interspace 1½" to left of sternum.	Recovery
39.	Riedel, 1897	M		Several ribs resected.	Died
40.	Mausell, Moulin, 1897	M	38	5th interspace.	Recovery
41.	Brentano, 1898	Ch.	7	5th cartilage resected.	Died
42.	Brentano, 1898	Ch.	7	5th cartilage resected.	Died
43.	Brentano, 1898	M	15	5th cartilage resected after incision in 5th interspace.	Died
44.	Brentano, 1898	M	31	Without resection.	Died
45.	Brentano, 1898	F	10	5th cartilage resected.	Recovery
46.	Robinson, 1898	M	4	1" removed over 5th cartilage.	Died
47.	Levestre, 1898	M	22	4th interspace, rib resec. for db. empyema; pleural inf. at first operation.	Died
48.	Ljunggren, 1898	M	24	5th interspace 2 cm. inside below nipple to 6 cm. outside. 5th rib resec. 2 wks. later.	Recovery
49.	Lilienthal, 1899	M	15	Resection 4th and 5th cartilages.	Recovery
50.	Bexman, 1899			2nd interspace.	Recovery
51.	Allingham, Ogle, 1900	M	26	Resection 5th rib cartilage.	Died
52.	Connor and Stimson			5th interspace rt. pleura opened.	Died

CASES OF PURULENT PERICARDITIS IN THE LITERATURE

(Continued)

	Author	Sex	Age	Incision	End-result
				53-74 Compiled by Eliot	
53.	Reichard, 1900	?	?	3rd cartilage resected.	Recovery
54.	Reichard, 1900	?	?	4th cartilage resected.	Recovery
55.	Mintz, W., 1904	M	12	5th cart. resected. Then along lower border of 7th rib.	Recovery
56. 57. 58.	Allingham, 1904			Mentions 3 of his cases in discussion with Coutts-Rowlands. Details missing; to appear later.	
59.	Coutts and Rowlands, 1904	Ch.	2½	5th left cart. 2nd op. resec. 5th rib in post. axillary line. Drainage good. Measles and pneum. caused death.	Died
60.	Coutts and Rowlands, 1904	M	1 yr. 8 mos.	Resection 7th cart. and gladiolus.	Died
61.	Kiliani, 1907	M	22	4th rib resected.	Recovery
62.	Geggenbach, 1906	M	8	5th rib resected 1½" to left of sternum. Pulmonary abscess.	Died
63.	J. A. Scott, 1904	M	36	5th interspace.	Recovery, but heart palp.
64.	Peters, 1903	M	7	4th interspace ¾" left of sternum; 5th right interspace; 5th left interspace, pleura opened; 4th interspace ¾" left of sternum, pus (staphylo. aureus found). Puncture at angle of left scapula drew pus. Under ether 30 oz. evacuated with rib resection.	Recovery
65.	Latham, Pendlebury, 1903	M	53	Allingham's method and additional exposure by res. sternal end 6th c.	Recovery
66.	Hall, 1908	F	38	Puerperal sepsis, db. pneumonia. 4th left cart. resected. Sup. peritonitis.	Died
67.	Sibley, 1903	M	16	6th rib res. outside nipple line.	Died
68.	Gluck, 1907	M	9	Subperiosteal res. 5th rib from point opp. mammary line 4 cm. ext.	Died
69.	Gluck, 1907	M	56	Same as 68.	Recovery
70.	Steward, Garrod, 1907	F	5	Rib. res. for empyema. Pericardium opened through empyemic cavity.	Recovery
71.	Godlee, 1907	Boy		Anterior incision. Complications: abscess of elbow and brain. Died 1 yr. later of rec. brain abs.	Recovery
72.	Pendlebury, 1904	M	31	6th and 7th cartilages.	Recovery
73.	Pendlebury, 1904	M	12	7th cartilage.	Died
74.	Eliot, 1909	F	19	Preg. 2 mos. miscar. 5th cart. res. 1" l. of sternum.	Recovered
				75-86 Compiled by Rhodes	
75.	Harrigan, 1913	F	11	Incision 4" beg. at. left sternal border, obliquely down and out, crossing left costal c. at its center 1½" of l. 5th rib and c. removed. (Staphylococci.)	Died
76.	Eiselsberg, ?	?	?	5th rib res. fistual open 9 wks.	Recovered
77.	Imerwol	M	9	Pleurotomy, l. 5th space axillary line; pericardium opened by orifice.	Recovery
78.	Imerwol	M	10	5th space 1 cm. from left s. toward axillary line.	Recovery
79.	Tallqvist	F	40	5th rib resected.	Recovery
80.	v. Wistinghausen			5th rib. res.; death from heart tamponade sev. days later.	Died
81.	Piradoff	M	26	5th and 6th ribs resected.	Recovery
82.	Blake			Pericardium opened in axillary line.	Died
83.	Wille	M	4	Incision with base along left side inc. 5th and 6th ribs subperios. res.	Recovered
84.	Rhodes, 1914	M	26	Hockey stick incision starting from 3rd costal at left sternal margin, down, curving outward along 7th rib. 4th, 5th cart. and portions of ribs resected subperiosteally. Pt. died 8 days after op.	Died

CASES OF PURULENT PERICARDITIS IN THE LITERATURE

(Continued)

	Author	Sex	Age	Incision	End-result
85.	Rhodes, 1914	M	28	Incision 6" parallel to l. border of sternum and costal margin of left ribs, ending internal to nipple. Periosteum of 6th rib injected with cocaine, stripped off. 2" of rib resected starting ½" from sternum. Pericardium stitched to anterior chest wall. Gutta percha drains.	Recovery
				86-101 Compiled by Pool.	
86.	Barie and Lebert	M	31	Pericardiotomy.	Died
87.	Boidin	?	?	Punctured the pericardium.	Died
88.	Robey	M	32	Operation.	Died
89.	Robey	?		Punct. 4th interspace, r. 4th and 5th cartilages resected.	Recovery
90.	Davis	M	11	4th and 5th costal cartilages.	Recovery
91.	Williamson	M	40	5th rib cartilage removed subchondrally.	Died
92.	Camac and Pool	M	47	Thoracotomy for empyema 6th cartilage.	Died
93.	Guillermo and Montaya	M	8	Inc. 5 cm. from sternal edge along 5th costal cartilage.	Recovery
94.	Jones	M	38	7th cort. cart. and middle of sternum. 7th, 6th, and 5th cart. and half width of sternum removed.	Recovered
95.	Klose	Soldiers		Extrapleural pericardiotomy using Renn's costo-xiphoid incision.	Recovered
96.	"	Bullet			Died
97.	"	Wounds			Died
98.	"	Purulent Pericarditis			Died
99.	Crabtree, 1918	Soldier		1) 2½" to left of sternum bet. 4th and 5th ribs. 2) 8" inc. to left, curving along 6th rib. 4th and 5th cart. and ribs resected. ½" of left margin of sternum removed.	Recovery (Lung Collapsed)
100.	Noble and Vine, 1919	M		5th rib and cartilage from mid. and nipple line. 5th cartilage resected.	Recovery
101.	Pool, 1921	M	9	After pneumonia. Hockey-stick incision intercostal thoracotomy for empyema.	Recovery
				102-115 Compiled by Williamson	
102.	White, M., 1925	M	7	5th cart. resected and adj. portion of sternum.	Recovery
103.	Durand, Wertheimer	M	37	7th costal cart. and lower portion of sternum.	Recovery
104.	Whittemore	M	12	Suction and drainage inc. 5th interspace.	Recovery
105.	Hilse	M	19	Ollier's method.	Died
106.	Hilse	M	27	Renn's method (adhesions remained).	Recovery
107.	Gamberini	M	7	Section of sternum removed 2nd op. resection, post. and ant. drainage necessary.	Recovery
108.	Apfel	M	12	4th cart. and l. border of sternum.	Died
109.	Jopson	M	16	?	Recovery
110.	Muller	M	Ad.	?	Died
111.	Muniaquiria and Zeno	M	4	Pericardiotomy.	Recovery
112.	Hall and Townrow	M	17	5th rib and empyema drained.	Recovery
113.	Wood, Bradley	M	4¾	Resected 3rd rib. Opened wound several times.	Recovery
114.	Adshead	M	22	Resected 5th rib and cartilage. Inc. abscess post. bet. base of lungs and dome of diaphragm.	Recovery
				115-117 Compiled by Peterson	
115.	Peterson	M	1	Along left margin of sternum and out over the 5th cost. cartilage. Cart. resected. Pus forced out under great pressure. Lung signs increased; baby died 36 hrs. after op.	Died
116.	Peterson	F	3½	6th costal cartilage resec.	Recovery
117.	Peterson		14	1) 7.6 cm. of 8th rib removed in post. axil. line. Lg. amt. pus evac. 2) 6th and 7th left costal cart. removed.	Recovery

CASES OF PURULENT PERICARDITIS IN THE LITERATURE

(Continued)

	Author	Sex	Age	Incision	End-result
				118-127 Compiled by Winslow and Shipley	
118.	Winslow and Shipley, 1924	M	4	Transsternal route. 4″ incision upward from apex of ensiform cart. in midline of chest. Inc. deepened to sternum, which was trephined with a Hudson burr.	Recovery
119.	Winslow and Shipley, 1925	M	18	Trephine opening in lower end of mid-sternum.	Recovery
120.	Winslow and Shipley, 1925	M	19	7th rib res. below inf. angle of scapula. Trephining of stern.	Died
121.	Winslow and Shipley	F	10	Transsternal route.	Died
122.	Winslow and Shipley	M	23	5th, 6th, 7th cart. res. just lateral to sternum.	Died
123.	Winslow and Shipley	F	9	Incision parallel with costal margin on l. side 10 cm. obliquely downward and to left from the junc. of ensiform app. with second portion of stern. 5 cm. of 5th, 6th, and 7th costal cart. removed close to sternum.	Recovery
124.	Winslow and Shipley	F	12	Same as 123.	Recovery
125.	Winslow and Shipley	M	22	5-in. incision in r. 5th interspace.	Recovery
126.	Winslow and Shipley	F	8	1 in. left 5th costal cart. empyema.	Died
127.	Winslow and Shipley, 1918	M	23	5th cart. res. to r. of stern.	Recovery
				128-133 Compiled by White	
128.	White, 1929	F	8	Inc. to l. of stern. border in 4th interspace, curving outward along 7th costal cart. 5th and 6th cost. cart. cut away.	Recovery
129.	White, 1922	M	9	Inc. as 128. 4th cart. 4th and 5th ribs resected.	Died
130.	White, 1922	F	6	R. pleu. cavity opened. Pericardium opened with cautery.	Recovery
131.	White, 1925	F	23	Converted into empyema, rib resection; cigarette drains.	Recovery
132.	White, 1926	M	24	Stab wound, rib res. 7th intercostal cartilage.	Recovery
133.	White, 1925	M	7	Rib resection, 5th.	Recovered
				134-152 Compiled by Truesdale	
134.	Ramond and Weill-Spire	M	23	Xiphoid-sternal. Diaphragm incised.	Recovered
135.	Albrecht	M	23	Costoxiphoid incision.	Recovered
136.	Albrecht	M	19	Recurrent acute pericarditis; bullet lodged behind sternum in lung. Rib resections.	Recovered
137.	von Eiselsberg	M	?	4th rib resected.	Recovered
138.	von Eiselsberg	M	19	5th cartilage resected.	Recovered
139.	A. J. and A. S. Welch	F	30	Transsternal route; after miscarriage.	Died
140.	Harrington	M	27	R. empyema, l. pleural effusion. Resected 7th rib in post. axil. line for empyema. Ant. pericardiotomy, resected 6th cart.	Recovered
141.	Harloe	F	6½	Incision 5th l. interspace.	Died
142.	Harloe	M	31	Same as 141.	Recovered
143.	Thomas	F	4 yrs. 10 mos.	Allingham's incision. 2nd incision done. 8th rib resected in post. axil. line.	Recovered
144.	Arntzenius	F	11	Trap-door incision. Staphylococcus aureus.	Died 4 wks. after op.
145.	Loucks	M	19	4th rib and cart. resected. Thoracotomy for empyema (8th rib res.).	Recovered
146.	Loucks	M	30	Rib. res. in midaxillary and postaxillary line. Lung perforated. Died of TB abscesses.	Died
147.	Brenizer	F	20	Puncture first. Then resec. of 5th and 6th cart. Patient died of abscess formed at punc. site.	Died
148.	Grimault	M	30	Resected 5th and 6th cart.	Recovered
149.	Garot	Child	4	?	Died
150.	Cairo	?	?	Trans-diaphragmatic sub-xiphoid puncture of pericardium.	Recovered
151.	Miller	M	8	5th and 6th ribs resected. 6	Recovered
152.	MacDonald	M	8½	Incision just to left of sternum intercostal (pneumococcal).	Recovered

Discussion

Dr. Fred B. Lund, Boston, Mass.: If you will read in Galen's "Anatomical Dissections," you will find that he has made a study of the peritoneum, and described it beautifully, as well as the pleurae and pericardium. He noticed that there was a place back of the sternum where the pleura didn't come together in the midline so that the pericardium could be opened without damaging the pleura. He had a case of a boy who had a blow over the sternum resulting in suppuration, and osteomyelitis of the sternum. He went in carefully avoiding the folds of the pleura, going in on the midline, and took out a large piece of the sternum, opening the pericardium and seeing the heart beat. He removed a large necrotic piece of the sternum and the child got well.

When I was a house officer, Dr. C. B. Porter operated on a young man, now an ophthalmologist in active practice, for pericarditis. I don't remember what incision was made, but he opened the pleura, and subsequently had to operate for empyema.

Dr. Truesdale: He made a parasternal incision.

Dr. Lund: The patient is well today, forty years after the operation. I never operated upon more than one case and that patient didn't live. I think this paper is a most brilliant and commendatory exposition of courage in the face of difficulties. When structures are pushed out of the way by a distended viscus or cavity, and you can take advantage of abnormal anatomy, you have done well.

Dr. Edward H. Risley, Waterville, Me.: Dr. Truesdale said nothing about aspiration previous to operation. I believe it is recognized that in young children especially, sometimes aspiration of an empyema will result in a cure without further operative procedure.

I can report one case of pericarditis in which this happened and I wonder how common the occurrence is. A boy of eleven years was brought in desperately ill, with an empyema on the right side following pneumonia. He was so ill that we resorted to aspiration twice before introducing an intracostal catheter and had further suction drainage. The empyema cleared up.

On the twelfth day he began to develop a rise in temperature and pulse, and had difficulty in respiration, and the x-ray showed an increase in the heart area. We felt within another twenty-four hours that he had a pericarditis with effusion. This was aspirated in the fifth space about an inch and a quarter from the midsternal line and to the left, and 200 cc. of a cloudy fluid was withdrawn which showed under the microscope definite formation of pus cells and staphylococcus growth. This immediately relieved him of his dyspnea and he made an uninterrupted recovery without further operative procedure.

This is the only opportunity I have had to aspirate early and have this fortunate result. I should like to know if Dr. Truesdale would advocate that as a routine procedure or whether he has had other experience with it.

Dr. Truesdale: I congratulate Dr. Risley on making an early diagnosis. Several writers state that in the suppurative type of pericarditis, aspiration fails invariably. If the exudate is serous, it can be aspirated successfully, but the frank purulent exudate in the pericardium is not cured by aspiration.

THE PROBABILITY OF DYING FROM ORGANIC HEART DISEASE

Out of every thousand white males born, over two hundred will eventually die of heart disease under present conditions of mortality; that is, approximately one in five. The chances (at birth) are in fact greater of eventually dying from heart disease, than from any other single cause, and even if we add together the chances of dying from two such important causes as tuberculosis and cancer, the sum for these two is actually less than the chances of dying from heart disease. For, according to mortality conditions as of 1930, the probability, at birth, of eventually dying from tuberculosis, among white males, is 43 per thousand, from cancer, 90 per thousand; so that the chances of eventually dying from either one or the other of these two causes are 133 per thousand, and less than the chances of dying from "heart disease,"* namely, 203 per thousand. The corresponding probability figures for while females are tuberculosis 36, cancer 122, summing up to 158 per thousand, as against 220 per thousand for heart disease.

At successive ages of life, the probability of dying from heart disease increases steadily among white persons until the neighborhood of the eightieth year of life, after which it decreases again.

Another interesting feature is the fact that the probability of dying from heart disease, in the white population at any rate, is greater for females than for males. This is consistently the case in the three calendar periods examined. The difference is not very great and is probably due, in whole or in part, to the familiar fact that women are longer lived than men and that, therefore, there must be a relative predominance of deaths among the former from causes characteristic of the older ages; and among these, organic heart disease is preëminent.

In this study the classification organic heart disease is such as to exclude angina pectoris. —*Bulletin of the Metropolitan Life Insurance Company.*

*For the purpcse of this article, the causes comprised under the term "heart disease" are those of International Classification of Causes of Death, Code Number 79 of the 1910 classification, in dealing with mortality of 1920; and Code Number 90 of the 1920 classification, in dealing with mortality of 1925 and 1930. These two combinations are not quite strictly comparable, but differ only with regard to a few relatively minor subclasses.

THE ROLE OF PSYCHIC LOAD IN RECURRENT ATTACKS OF GASTRODUODENAL ULCERS

BY MOSES EINHORN, M.D.*

THE medical profession has long been acquainted with the relationship between disturbances of the nervous system and the digestive function. Emotional disturbances of sudden occurrence, as anger and grief, or disturbances of a lesser degree, but of long duration, as domestic trouble, business stress or fear, have a tendency to affect the digestive apparatus. Worry, anxiety, irritation or undue fatigue, all exaggerate gastric symptoms. Extreme happiness, as well as depression, also react unfavorably on the gastro-intestinal tract.

In relation to the above, Kaufmann[1] has stressed the importance of the psychic element in the treatment of peptic ulcer. He points out that psychic influences such as prolonged mental strain, shock and emotion are accessory factors, which play a causative rôle in the development of ulcers, and react especially in certain types of individuals. He states that at first this condition is noted as a simple pylorospasm, associated with hyperacidity, which, when prolonged, may finally produce an erosion and then an ulcer.

Taylor[2] refers to psychic load as a factor, and cites a number of cases in which psychic elements undoubtedly play an important rôle. A salesman has an attack each time his territory is changed. Another, a bootlegger, has symptoms which are very difficult to control. Several are of the rapidly advancing "Young America" type, who can scarcely wait until they have accumulated sufficient money to retire. One patient had his first symptom after the death of a relative. Taylor is of the opinion that psychic wear and tear is apparently communicated to the digestive tract as a definite trauma, which probably finds pathological expression in the blood supply at the site of the ulcer.

Fitz[3] relates his observations on psychic influences, and states that the psychic factor is apparent in both sexes. One woman was symptom-free except when she quarreled with her husband. Another, a professional public speaker, had prostrating symptoms whenever she developed stage fright at addressing an audience. Business worries, also, induced symptoms quite regularly in both sexes.

Rogers and Hardt[4] have shown that psychic influences are capable of interfering with the normal course of gastric rhythm. Monsarrat[5] is in agreement with this, since he is of the opinion that the alteration in gastric rhythm is the primary morbid condition which when persistent leads to the development of ulceration.

I have been particularly interested in the study of ulcers, and have had special opportunities for this study in all its phases, both here and abroad. In my observations and investigations I was impressed by the effect of cold, diet, trauma and psychic load on numerous ulcer patients, and the tendency of these factors to cause recurrent attacks. I have made a detailed analysis of the factors[6] which influence the production of recurrent attacks of gastroduodenal ulcers, and found, in this analysis, that psychic influences, such as anger, shock, emotion and business reverses exert a causative rôle in the development of recurrent attacks, psychic load representing seven per cent of all these recurrences.

I am citing below several cases typical of a group in which psychic influences have played a rôle in the production or recurrent attack of ulcers.

(1.) A. A., aged forty-five years. Gastric complaint for ten years. Recurrences, on and off, at irregular intervals. *Symptoms:* Pain in the epigastric region, three hours after meals. Relieved by food and bicarbonate of soda. Night pain; heartburn, sour eructations. *Roentgen-ray findings:* Irregular duodenal bulb. No six-hour gastric retention. *Ewald test:* F-Hcl: 98; total acid: 118. *Occupation:* Singer. Nervous, neurasthenic patient; reflexes greatly increased. A detailed clinical history revealed that recurrent attacks usually occurred during the fall and winter, while the patient was engaged in rendering concerts. During the summer, when free from engagements, or in any other period while not engaged, there were no recurrences.

(2.) J. G., aged thirty-three years. Gastric complaint for eight years. Recurrences mainly in the fall. *Symptoms:* Pain in the epigastric region, pressing in character, three to four hours after meals. Relieved by food and bicarbonate. Night pain, at 2 A.M. No vomiting; heartburn. *Roentgen-ray findings:* Irregular duodenal bulb. No six-hour gastric retention. *Ewald test:* F-Hcl: 72; total acid: 118. *Occupation:* Merchant. A detailed clinical history revealed that the patient's loss of a huge sum of money, due to the failure of a bank, caused a recurrence. This attack occurred during the winter although the patient had never previously had an attack at this time of the year.

(3.) R. M., aged fifty-one years. Gastric complaint for three years. Recurrences intermittently. *Symptoms:* Pain in the epigastric region, three to four hours after meals. Relieved by food and rest. Night pain, at 3 A.M. No vomiting. *Roentgen-ray findings:* Bulbus duodeni shows persistent defect of the inner and outer border. No six-hour gastric retention. *Ewald test:* F-Hcl: 53; total acid: 64. *Occupation:* Races. Nervous and neurasthenic patient. A detailed clinical history revealed that recurrent attacks were due mainly to extreme excitement caused by excessive gains or heavy losses on the Saratoga race tracks.

*Einhorn—For record and address of author see "This Week's Issue," page 708.

(4.) I. M., aged thirty-eight years. Gastric complaint for three years. Recurrences mainly in the fall, and immediately following a cold. *Symptoms:* Pain in the epigastric region, three to four hours after meals. Relieved by food and bicarbonate of soda. Night pain, at 4 A.M. Heartburn, sour eructations. *Roentgen-ray findings:* Bulbus duodeni does not fill completely, and shows irregularity of the inner border. Small six-hour gastric retention. *Ewald test:* H-Hcl: 32; total acid: 64. Extremely nervous and sensitive patient. Under observation for a few years, with recurrences mainly in the fall, usually after a cold. A detailed clinical history revealed that on this particular occasion the patient arrived home and learned of the sudden death of a member of the family. A recurrent attack followed. This attack occurred in the summer, although the patient never previously had an attack at this time of the year.

(5.) R. F., aged twenty-six years. Gastric complaint for three years. Recurrences at irregular intervals, with no seasonal relationship. *Symptoms:* Pain, in the epigastric region, a few hours after meals. Relieved by food. Night pain; heartburn; sour eructations. *Roentgen-ray findings:* Irregular duodenal bulb. No six-hour gastric retention. *Ewald test:* F-Hcl: 32; total acid: 64. A detailed clinical history revealed that after a family dispute or argument, the patient became nervous, had palpitations, and thereafter a recurrence.

(6.) J. S., aged thirty-nine years. Gastric complaint for a few years. Recurrences on and off, in the spring or fall, immediately following a cold. *Symptoms:* Pain in the epigastric region, two to three hours after meals. Relieved by food and bicarbonate of soda. Night pain; heartburn; sour eructations. *Roentgen-ray findings:* Irregular duodenal bulb. Slight six-hour gastric retention. *Ewald test:* F-Hcl: 27; total acid: 63. A detailed clinical history revealed that a recurrent attack was induced by sudden fluctuations in the Wall Street market, in which the patient was an investor. This attack occurred in the summer, although the patient previously did not have an attack at this time of the year.

As mentioned above, stage fright on appearing before an audience, extreme excitement caused by excessive losses on the race track, shock due to a sudden death in the family, and extreme happiness or depression attributed to fluctuations in the Wall Street market; these are but a few of the psychic elements which affect the production of recurrent attacks of ulcers.

It has been definitely established that duodenal ulcers usually occur in individuals between 20 and 30 years of age, and gastric ulcers between 40 and 50 years of age. The initial attack is ordinarily of short duration, followed by a long period of dormancy. For several years there may be no recurrence, but as the years progress, the period of dormancy is gradually diminished, and the attacks become more frequent.

Recently[6], I made an extensive study of the seasonal incidence of ulcers and the various factors which influence the production of recurrent attacks. I found that ulcers recur during specific seasons of the year, particularly in the spring and fall, and that factors such as cold, diet, pressure, psychic load, etc., have a tendency to exaggerate ulcer symptoms and indirectly cause recurrences.

The question arises as to the manner in which these factors give rise to an ulcer attack. In a separate article[7], I am advancing my new conception of the formation of recurrent attacks of ulcers, explaining the rôle which the various factors play in their production. I shall give here merely a summary, to point out the part which psychic load plays in these attacks.

In an ulcer attack it is important to consider, first, the forces which cause the attack; secondly, the forces which resist the attack; and thirdly, the neutral zone or period of dormancy. Figuratively, an ulcer may be characterized as follows:

A is the attack.

F are the unknown forces which stimulate the attack.

R is the resistance and includes the constitution, habits, of life, etc.

D is the period of dormancy.

A will result if (1) R is smaller than F.
(2) F is larger than R.

D will result if (1) R is larger than F.
(2) F is smaller than R.
(3) R is equal to F.

EXPLANATION

In a distinct group of individuals, certain unknown forces cause pathological changes in the blood supply of selective regions of the stomach and duodenum producing first an erosion and finally advancing to the stage of an ulcer. These changes give rise to the characteristic symptoms of an ulcer, namely, the typical pain, belching, heartburn, etc. Meanwhile, other forces in the body tend to resist the attack, gradually promoting the healing of the affected area. The symptoms then disappear, and a period of dormancy follows, when the patient is free from any complaint. This period may be likened to the neutral zone—that which lies between the contending forces. When the attacking forces are increased or the resistance decreased, the neutral zone is disturbed, and the attack stimulated, but when the attacking forces are decreased and the resistance increased, the neutral zone maintains its equilibrium, and the dormant period, or period of intermission is, as a result, prolonged.

In my study of factors influencing the production of recurrences of gastroduodenal ulcers, mentioned heretofore, colds were found to be the most important, being present in 57 per cent of all recurrences. Analysis of the degree and intensity of the cold revealed that the mild cold constituted the highest percentage, the more severe cold, a moderate percentage, and the acute condition, such as grippe and influenza, comprised the remaining small percentage.

Diet as a causative factor was found in only

9 per cent of the recurrent attacks, a surprisingly low percentage in view of the fact that the treatment of ulcer is essentially dietetic.

External pressure[9] was represented in only 3 per cent of the gastroduodenal recurrences.

Psychic influences, such as anger, grief, shock, emotion and business reverses play a causative rôle in the development and recurrent attack of ulcers, the percentage being 7 per cent. Worry, fear, anxiety, irritation or undue fatigue are all known as accessory factors in producing and perpetuating ulcer symptoms, and are grouped under the definite term of psychic trauma.

COMMENTS

Inasmuch as neither the etiological nor the defensive forces, but merely the disturbing factors involved in the production of ulcer recurrences are known, in treating the patient, we must endeavor to ascertain and eliminate those factors which may be instrumental in promoting the attack.

When, for example, colds are the causative factor, all sources of focal infection, such as diseased tonsils and teeth are removed; if diet is the disturbing factor, foods which disagree with the patient are eliminated, and a suitable diet prescribed. In like manner, the psychic element should not be overlooked, and in outlining the treatment, not alone the ulcer, but the entire patient requires consideration. His mode of living, environment, occupation, etc., should be carefully investigated, and all harmful psychic influences eradicated. A long period of mental and physical rest, free from worry or anxiety is absolutely essential for the patient. He should be placed on a simple diet, with sufficient alkali to overcome his symptoms, and thereafter plan to live a well-regulated normal life for months or even years. Faith and confidence in his physician will make the patient more comfortable, and incidentally hasten his recovery. The importance of visiting the physician in the spring and fall, to prevent recurrences, should also be stressed.

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REPORT OF A CASE OF ACUTE HYDRAMNION TREATED BY ABDOMINAL PUNCTURE

BY BENJAMIN PARVEY, M.D.*

IT is said that Puzos[1, 3], in 1759, for the first time observed the anomaly of excessive amniotic fluid in pregnancy, and gave to it the name Hydramnion. Sédillot[2, 3] in 1797, gave the first description of acute hydramnion. Authorities do not agree as to the exact limit of the normal amount of amniotic fluid in pregnancy, but in general consider any amount above two liters abnormal. It is believed that the amnion is formed from the external layer of the blastodermic membrane and contains a sero-albuminous fluid, which constantly increases to accommodate growth and the movements of the fetus[4]. Küstner[5] noted as much as fifteen liters in one case in the fifth month of pregnancy, and Sallinger[6] reported a case with the same amount of fluid at the end of the seventh month. Werth[7] and Forssell[8] found as much as fifteen to twenty liters in their cases. In Schneider's[8] case, as much as thirty liters was obtained in the fifth month of pregnancy. Wilson[4] obtained twenty-eight liters in a case with a large fetus and one liter in a case with a small fetus. The incidence of hydramnion has varied in the reports of different clinics; an average estimate suggests about one case in every four hundred and fifty deliveries. Acute hydramnion is considered rare by Chambrelent[9]. Bequain[10] reported one case of acute hydramnion in twelve thousand births. Lion[10] in a review of six hundred and twenty-three reported cases of hydramnion found only eight that were acute. Plauchu[11] believes that there are two forms of acute hydramnion, one that appears in the early months of pregnancy, is generally associated with twin pregnancy and results in abortion, and one that develops in the later months.

The etiology and origin of hydramnion are obscure. It may arise from the mother, the fetus, or the amnion itself. Many investigators believe that a chief etiological factor is an inflammatory condition of the amnion which results in an increased exudation of fluid. Schroeder[12] has shown that the fluid can transude from the amnion into the cavity of the fetal sac. Wilson[13] states that the hydramnion appears to occur nearly as frequently in the separate ova as

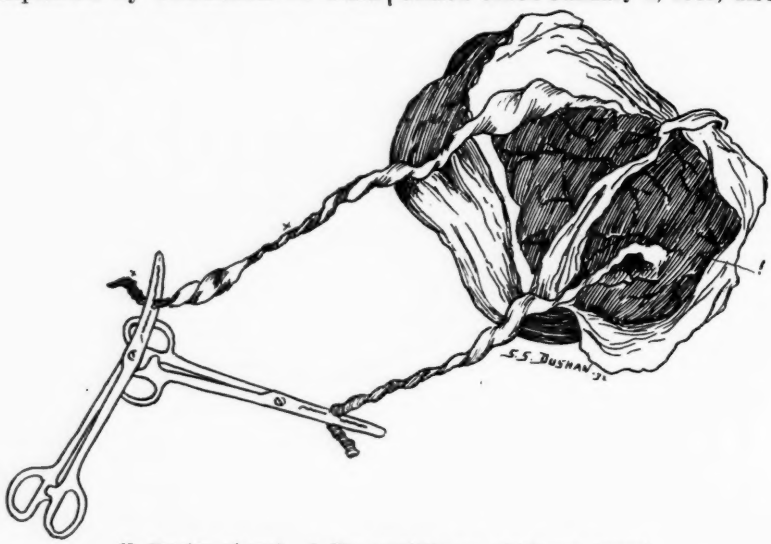
*Parvey—For record and address of author see "This Week's Issue," page 708.

in the single ovum twin pregnancy. He suggests that hydramnion in twin pregnancy is related to the greater rapidity of growth of one fetus, which leads to a greater absorption of fluid by that fetus, and under rare circumstances results in hydramnion. Another theory holds that circulatory disturbances of the heart, kidneys, and visceral systems may lead to edema of the placenta, resulting in an increase of transudate into the amniotic cavity. Bruno Wolff[14] has experimentally demonstrated that by nephrectomizing pregnant rabbits he could produce acute hydramnion, which was followed by an increased renal activity on the part of the fetus. Scheib[15] believes that in circulatory failure the fluid transudes the umbilical vein. The relation of obstruction of the circulation in the umbilical vein to hydramnion was shown by Sallinger[6], who demonstrated that such obstructions were accompanied by exudation of fluid from the external surface of the umbilical cord and from the fetal surface of the placenta. Franqué[16] noted that obliterative changes in the coats of the arteries of the chorionic villi may lead to a similar result. Placental infarction was suggested by Orth[17], and torsion of the cord by Fehling[18] as the probable mechanism in this condition. Opitz[19] observed cirrhotic changes in the liver in all of his cases.

Very little has been proposed for the treatment of hydramnion. Schatz[20], in 1882, proposed an abdominal tap of the amniotic fluid for relieving excessive intrauterine pressure, but he never attempted this operation himself. Many theoretical objections to this procedure were raised by Seitz[21] and Poeck[22]. Some of these are the possibilities of injury to the child, disturbance of the circulation in the placenta, rupture of the uterus, and infection. The procedure was nevertheless tried and reported on by Forssell[3], Henkel[23, 24] and Wormser[25]. These authors state that Bumm, in 1900, was the first clinician to have attempted the procedure.

F. L., para IV, a housewife of 29 years of age, was first seen on August 20, 1931. She complained of nausea, distress after eating, headaches and insomnia.

Her first pregnancy occurred seven years ago, natal complications. She aborted her second pregnancy two years later in the third month. The cause was unknown. A third pregnancy three years ago was terminated artificially because of severe pernicious vomiting, which started in the third month. The fourth and present pregnancy began July 6, 1931. Severe pernicious vomiting appeared in the second month. This resulted in acidosis and the loss of considerable weight. Treatment with intravenous infusion of a 20 per cent glucose salt solution with insulin was successful. Her condition became normal, and she resumed her daily routine. There were no symptoms until December 29, 1931, when it was noted that her abdomen suddenly began to swell and reached such a degree of distention within a few days that she could not retain food or fluids. This was followed by suppression of urine, a fall in blood pressure, and an elevation in pulse. A diagnosis of hydramnion was made. X-ray of the abdomen taken January 2, 1932, disclosed a twin preg-



X Torsion of cord. ! The amniotic sac which was tapped.

nancy. Her condition became progressively worse. Urinalysis on January 6, 1932 showed a large trace of acetone and albumin, and a large amount of bile. The blood sugar was 50 mg. per 100 cc. of blood; the blood Wassermann was negative. She was given 750 cc. of a 20 per cent glucose saline intravenous infusion and sent to the hospital. An able consultant advised the immediate termination of the pregnancy, but the patient refused.

On January 9, 1932 an abdominal paracentesis was performed under the usual surgical asepsis with local anesthesia. A lumbar puncture needle was inserted over the most fluctuating point of the abdomen, about two inches below the umbilicus. Seventy ounces of the amniotic fluid were removed slowly at a rate of one hundred and forty to one hundred and sixty drops per minute, the entire procedure requiring approximately three hours. Immediate relief followed. She excreted thirty ounces of urine in the next twenty-four hours, and was able to take food with relish. Approximately forty-eight hours later, however, the abdomen again began to swell and the same symptoms gradually returned. A second tapping was performed on January 15, 1932. Twenty-four hours later the patient went into active labor. The cervix was fully dilated. The bag of water protruding through the cervix was ruptured, and about three liters of fluid gushed out. This

was followed by the birth of a macerated female fetus, which on examination revealed a cord twisted in two places. This fetus weighed two pounds and thirteen ounces. The second female fetus was born several minutes later, weighing one pound and eight ounces. It survived for three and one-half hours. The second amniotic sac contained one quart of fluid. An autopsy was performed which showed an intense congestion of all organs. The patient made an uneventful recovery.

DISCUSSION

Although the patient aborted, the abdominal tap relieved the mother of such distressing symptoms as vomiting, dyspnea, pain and suppression of urine. The cause of hydramnion in this case being due to a twisted cord, the procedure could not be expected to offer more than temporary relief. In some cases of hydramnion not related to mechanical obstruction of this type, it is conceivable that repeated tapping may permit the normal course of pregnancy, and for this reason the procedure should be given consideration. It further will enable the determination of the position of the fetus.

After the first abdominal tap, in which two liters of amniotic fluid were removed, there was still evidence of more fluid. As a rule hydramnion is limited to a single amnion. The reason for the cessation in the flow was not entirely explained, but as noted on delivery there were two amniotic sacs, and it was likely that the first tapping merely drained the fluid from the amnion contained in the viable fetus. If this were fully realized and if it were possible to map out both amnions, the abdominal tapping could be directed more intelligently.

Some authors have suggested that hydramnion may at times be caused by the urinary excretion of the fetus. Schaller[26] demonstrated that the fetal kidneys function only to a very slight extent during intrauterine life. Rissmann[27] has reported a case in which a fetus with congenital absence of both kidneys was born alive just before term, proving that the renal function is unnecessary to intrauterine life. The function of the fetal kidneys in the present paper was tested by injecting 1 cc. of phenolsulphthalein into the antecubital vein of the patient two hours prior to the second tapping. An effort was made to detect the dye in the amniotic fluid removed, by the usual method of alkalinization with 20 per cent NaOH, as for urine. The patient's urine showed a 50 per cent excretion at this time, and the amniotic fluid gave no evidence of the dye. This would appear to corroborate the opinion already expressed that urinary products of the fetus probably do not enter the amniotic cavity.

The available statistics on the outcome of hydramnion do not warrant the hopeless attitude in the treatment of the condition. Krahula[28] studied the outcome of a group of two hundred and seven cases of hydramnion deliveries in the Bonn Clinic, Germany, between the years 1877 and 1920. He stated that twenty-eight were born viable and left the hospital. Floris[29] studied a group of two hundred and thirty-six cases delivered at the Kermauner Clinic, Vienna, during the years 1910 to 1921. One hundred and eighty-three were born viable and left the hospital. Of a group of sixty-five that he was able to follow after delivery, seven died of infantile diseases, but the remaining fifty-eight were alive and normal. He felt that hydramnion deliveries, when born free of mental and physical anomalies, should be expected to do as well as other normal children.

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ARSPHENAMINE IN THE TREATMENT OF VINCENT'S ANGINA

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VINCENT'S angina is a sporadic infection of the oral mucous membranes and is frequently confused with subacute or chronic tonsillitis, diphtheria and syphilis. In the following case history Vincent's angina appeared as an aftermath of acute tonsillitis and obdurately resisted treatment for four weeks until arsphenamine was employed locally and intravenously.

Vincent's angina recently termed fusospirillosis was first described in 1893 by Rauchfuss who, however, did not associate the fusiform bacillus and the spirillum with the condition. In 1894 Plaut described the condition and a certain organism which Vincent maintained was not the fusiform bacillus, definitely associated with the disease. In 1896 Vincent described the disease and two organisms associated with it and in 1928 apparently established his claims to priority in the description of the bacillus fusiformis and the disease processes caused by it. Two hundred years before Vincent (1683) Leeuwenhoek, the early user of the microscope studied one of this group of organisms.

The fusiform bacillus and the spirillum may be present on the mucous membranes of healthy mouths and in examinations made of large groups as high as fifty per cent of those examined with no inflammation in the mouth showed the presence of the organism. The commonest site for these organisms is in the crypts of the tonsils, next on the gums especially about the lower molars, and occasionally they have been known to invade the middle ear, vagina and lungs. In diabetes, syphilis, the leukemias and tuberculosis the organisms grow easily on the oral mucous membranes and cause profuse gingivitis.

I recently found the organisms in the stomatitis of pellagra. In this patient mild oxidizing agents, hydrogen peroxide and sodium perborate washes cleared the condition. Again at the Massachusetts Reformatory for Women I have observed these organisms in the stomatitis of patients treated intensively for syphilis with mercurials alone. When these patients were taken off the mercurials and the arsphenamines substituted intravenously, the stomatitis subsided and the fusospirillosis disappeared.

The treatment of Vincent's angina has been varied and has undergone many changes. At the present time the use of mild oxidizing agents locally or the arsenicals locally or intravenously are preferred. One of the early reports in the literature on the use of salvarsan locally in the treatment of Vincent's angina was made in 1919 by Teuscher, a German physician, and the intravenous use of salvarsan in three cases was reported in 1920 by Young. Since then the use of salvarsan intravenously has been condemned as unwarranted especially during the years 1929 and 1930. However at the present time it seems to be the generally accepted treatment of most clinicians and is particularly advocated by Albray.

CASE REPORT. H., aged twelve, height 5'2½", weight 105 lbs., was an athletic, healthy schoolgirl who, except for occasional head colds, had had no serious illness since childhood. During the period from two to five years of age she had pertussis, varicella rubeola and scarlatina. At six years of age and again at twelve she was immunized with diphtheria toxin-antitoxin. Her teeth and gums were in excellent condition. Prior to the present infection her tonsils were small and there was no previous history of tonsillitis.

During the last few days of August while vacationing on Buzzards Bay, her older sister, her younger brother and finally she herself developed acute tonsillitis. Among the children in the summer colony there was an epidemic of sore throat. The patient's sister and brother were ill about forty-eight hours with temperatures of 101°F. to 103°F., slight prostration and injected throats without exudate.

On September third, the patient complained of slight soreness of the throat and general malaise, and on examination showed a few discrete spots on both tonsils with injection and swelling. Her temperature was normal and the condition appeared to be a typical mild follicular tonsillitis. The constitutional symptoms lasted three days. In the following ten days there was a change in the appearance of the exudate from the discrete yellowish patches to more numerous minute greyish yellow spots which gradually coalesced into a pseudomembrane.

In the middle of September, as the infection was not subsiding in spite of local applications, a culture and smear were taken. The bacteriologist reported the smear and culture negative for Klebs-Loeffler bacilli, rods and spirilli, but positive for staphylococci. A consultation was held with a laryngologist.

The general treatment had been rest, forced feedings, cod liver oil and tablets containing iron gr. 5, arsenic gr. 1/40, and strychnine gr. 1/60; locally, a succession of antiseptics was applied, beginning with iodine and glycerine equal parts, tincture of ferric chloride, argyrol 50%, silver nitrate 10%, sodium perborate 2% as an application and as a gargle. With each preparation a momentary improvement occurred, to be followed, however by a recrudescence. In the meantime the patient's general condition remained unimpaired. She had no fever and was without physical discomforts.

In the first week of October a smear from the scrapings of the tonsillar exudate made at the Leary Laboratory in Boston showed the presence of the fusiform bacillus and the spirillus associated with Vincent's angina. In view of the futility of the treatment to date and considering the specificity of arsphenamine for spirochetes, neoarsphenamine gram 0.3 was given intravenously and sulpharsphenamine gram 0.6, dissolved in grams 15 of glycerine, was applied locally every four hours. Within twenty-four hours, resolution began and in three days the

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tonsils were clear of membrane. Both tonsils had become smaller through erosion, the right tonsil showing a linear cicatrix, the left numerous pin head cicatrices.

Vincent's angina, sometimes called ulcerative tonsillitis, pseudomembranous angina or trench mouth, was formerly considered a rare disease, but it was very common during the World War among the soldiers. Vincent, a French physician, some years ago described the condition and found constantly associated with it two organisms, a spirillum and a fusiform bacillus, which appear in large numbers on the stained field to the exclusion of any great number of other organisms or of pus. The disease occurs most frequently in young persons and is generally associated with a debilitated state of health, carious teeth or irritative lesions of the mouth.

The characteristic lesions of Vincent's angina are greyish white patches sometimes separated by healthy tissue involving the tonsil, soft palate, pharynx or gums. A pseudomembrane formed by the necrosis of the superficial epithelium covers the patches. Removal of this membrane leaves an ulcerated area which bleeds freely and is soon covered by a new membrane. The ulcerations are occasionally very destructive, eroding the tonsil and invading healthy tissue.

The smear taken directly from the ulcer and stained with Loeffler's methylene blue, fuchsin or diluted formalin gentian-violet shows two organisms: one the fusiform bacillus, a slender rod four to eight microns long, pointed at both ends, sometimes curved and often lying in pairs end to end; the other, a spiral organism ten to twenty microns long with three to ten shallow irregular curves. At present these two anaerobic organisms are considered stages of the same organism and are classed under the spirochetes. These organisms cannot be considered of etiological significance unless they are found in considerable numbers and dominate the picture, as they are not infrequently found in small numbers in normal mouths. Differentially the disease may be distinguished from diphtheria or syphilis by examination of the smear.

The constitutional symptoms of Vincent's angina are those of a subacute tonsillitis, the temperature normal or elevated a few degrees, some discomfort in swallowing and mild submaxillary and cervical adenitis. The ulcer below the membrane may be single or multiple. The membrane may spread rapidly and the ulcer persist for weeks or months with recurrences. The disease runs an acute or subacute course and often becomes chronic; however, most attacks are mild, persisting for several days or weeks.

The treatment is of two types, local and general. Under local treatment many agents are used, such as tincture of iodine, sodium perborate, 10% solution of silver nitrate, 6% solution of zinc sulphate, 25% solution of chromic acid, 1% solution of copper sulphate and ethyl chloride applied to the lesions. General treatment consists of cod liver oil and tonics. For the specific action on the spirochetes, arsphenamine for similar preparations may be used intravenously and locally mixed with water or glycerine.

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URETHRITIS DUE TO TRICHOMONAS*

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DONNÉ[1], in 1836, made reference to trichomonas as the possible provocative factor in cases presenting an obdurate and persistent leukorrhea as their main symptom. Nearly one hundred years have passed since the publication of his article and in the meantime numerous papers have appeared both in this country and abroad which tend to confirm his view. Yet our present knowledge of the life history[2], morphology[2], mode of transmission[2] and pathogenicity[2] of this flagellated protozoan is still very meager.

It is generally agreed that the trichomonas vaginalis has a pyriform or spindle-shaped body with free flagella at the anterior pole, an undulating membrane extending backward to near the middle of the body, and an axostyle which projects from the posterior pole. There are four anterior flagella or three as some authorities maintain. Another states that the so-called undulating membrane is not a membrane but is a pseudopodium. The size varies, but it is usually larger than a polymorphonuclear leukocyte and smaller than an epithelial cell. It

*Read before the staff meeting of St. Francis Hospital, Miami Beach, Florida, April 3, 1932.

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moves quite rapidly with the jerky forward motion and counterclockwise rotation common to most trichomonads. Reproduction by binary fission is agreed on but the question of cyst formation has not been definitely decided.

Neither the source of the organism nor the mode of transmission is known. Some authorities believe this flagellate to be identical with the trichomonas found in the intestines, while others maintain that it is distinct from the other trichomonas found in man. According to Greenhill[3] it is unusual for a woman with trichomonas vaginitis to have trichomonas in the stools and when the organisms from such a woman are implanted into the vagina of a healthy woman the latter does not develop a vaginitis. The same authority states that the question of pathogenicity is largely an academic one, for clinical experience has shown that the disappearance of the parasite coincides with the disappearance of the symptoms in the majority of cases. Haussmann[4] and Varela[5] have made similar observations.

The organisms are detected by examination of a fresh drop of the secretion with the high power lens. They are not revealed by the ordinary stained smears.

In view of the frequency of trichomonas infestation in the female genital tract, the question naturally arises as to whether it is limited to the female or is also found in the male. In reviewing the literature one must come to the conclusion that the organism frequently invades the female genital tract, but that it is rare in the male.

OCCURRENCE IN THE URINE

Künstler[6], in 1883, discovered the trichomonas in freshly voided urine and Davis[7], in 1896, found the organisms in the urine and vagina of a woman suffering from nephritis and diabetes. Arnold[8], in 1914, and Rasmussen[9], in 1926, isolated the parasite from the urine of pregnant women. Other writers who have discovered the protozoan in the urine of women are Baatz[10], Seitz[11], Flaskamp[12], and Escomel[13].

Lewis and Carroll[14], in 1928, reported the case of a young married woman who had been ill for three weeks with chills, fever and pain in the left side. Pyelitis was suspected, but all findings were negative except after long centrifuging of the catheterized specimens of the urine. Later the organisms from the bladder urine and the vagina were also recovered. The patient was treated by pelvic lavage with two per cent silver nitrate daily, 1:2000 solution of silver nitrate bladder irrigations and diathermy treatment in the vagina. With the disappearance of the organisms the symptoms subsided.

Visher[15], in 1929, reported a somewhat similar case.

TRICHOMONAS IN THE MALE

Miura[16], in 1893, found trichomonas in the freshly voided urine of a 52 year old man, but examination of a catheterized specimen was negative.

Marchand[17], in 1894, was the first to find the parasite in the bladder. He reported the case of a 60 year old man who for 17 years had had an abscess which was probably tuberculous. The abscess ruptured, probably into the bladder, and the urine became cloudy. Examination revealed epithelial cells, erythrocytes, hyaline casts, bacteria and trichomonads. He also found the parasites in a catheterized specimen.

Dock[18], in 1896, reported the flagellate in the freshly voided urine of a 27 year old student who for 12 years had suffered with urgency, pain on urination and often hematuria, which was thought to be due to malaria.

Katsunuma[19], in 1924, reported the organism in the urine of a three year old boy. The prepuce was red and edematous. The mode of transmission in this case was thought to be by means of the fingers of a woman.

Dastidar[20], in 1925, examined 1000 specimens of urine. He found trichomonads in three male specimens and in one female specimen. He noted a mild urethritis with burning on urination which subsided as soon as the flagellates disappeared.

Capek[21], in 1927, reported two cases of trichomonas urethritis in the male. His first patient had lost weight and had had a cloudy, blood-stained urine for eight years. The discharge was purulent, contained many pus cells, many red blood cells, numerous trichomonads, many nonspecific bacteria and was rich in fibrin. The wife of this patient was examined and the trichomonas recovered from the vagina and urethra. The other patient had a similar discharge and in addition an acute epididymitis. Capek expressed the opinion that infections in the male are more frequent than the literature would indicate.

Riba and Perry[22], in 1929, reported two cases. One, a male, 28 years old, complained of urethral discharge and nocturia following intercourse. The prostate was normal in size, slightly irregular and infiltrated. The prostatic secretion was examined in the natural state and trichomonas found. The other case, a male, aged 61, colored, complained of a daily frequency and nocturia every two hours with slight burning on urination and a week later developed a purulent urethral discharge which on examination in its natural state contained a few motile trichomonas and many pus cells. The prostatic secretion contained many motile trichomonas, a large number of pus cells with clumps and a few motile spermatozoa.

Grimm[23], in 1930, reported five cases of trichomonas urethritis in the male. His first case

was a man, aged 34 years, married, who had had a gonorrheal urethritis four years before. There were no complications and he was thought to be cured. For the past year he had had a urethral discharge which had been treated but without improvement. Many trichomonads were found in the discharge from the urethra. Examination of the prostatic secretion was negative for the parasites. Trichomonads were isolated from the wife of the patient.

The second patient was a man of 25 years, single, who had never had gonorrhea. The last coitus was four months before. For two weeks there had been a urethral discharge, at first mucoid in character and later purulent, with slight burning at the end of urination, and painful erections.

The next case was a man of 42 years, single, with a history of having had gonorrhea three years before. For the past three months he had been treated for a nonspecific urethritis without improvement.

The fourth patient was a single man of 25 years, who had had a gonorrheal urethritis and epididymitis one and one-half years before. This was thought to be cured and a short time later a clear secretion appeared which soon disappeared and then returned again. For the past month he had had burning at the meatus and for one week a purulent urethral discharge.

The last patient was a man of 29 years, single, with a history of having had gonorrhea one year before. Intercourse had occurred three weeks ago and for the past two weeks there was a purulent urethral discharge with burning during and after urination. The discharge was increased after taking alcoholic drinks. These five cases were seen by Grimm in a period of five months.

Riba[24], in 1931, reported two additional cases. One case, a male, aged 36, with the complaint of a urethral discharge, itching and a burning sensation in the penis for eight days had trichomonas in both the fresh urethral and prostatic secretions.

The other patient, a male Negro, aged 36, complained of itching on the ventral surface of the glans penis, a urethral discharge, burning on urination, urinary distress after intercourse, and nocturia once or twice for the past six months. Both the urethral discharge and the prostatic secretion contained motile trichomonads.

Rosenthal[25], also in 1931, reported one case, a male, aged 72, who had recently had a laparotomy for the operative treatment of cholelithiasis, complained of painful micturition, with suprapubic pain and scanty urine. Microscopic examination of the urine revealed many motile trichomonads. Rectal examination revealed a moderate degree of enlargement of the prostate, but there was no tenderness. Examination of the prostatic secretion was negative for parasites. On reëxamination of the urine a degenerate form of trichomonas was found in a granular cast and Rosenthal mentioned that this finding drew attention to the possibility of the organism ascending to the renal tissue itself.

I wish to put on record another case of trichomonas urethritis in the male, complicated with balanitis and trichomonas prostatovesiculitis.

CASE REPORT: G. P., a white male, aged 21, was seen at the office with the complaint of a urethral discharge, burning on urination for the past eight days and nocturia eight to ten times for the past five days. He stated that he had not had gonorrhea previously. The pulse was 88 per minute and the temperature 100.4° F. The general physical examination was negative except for the genitourinary region. The prepuce was long and markedly swollen and could not be retracted over the glans. The glans and prepuce were covered with dried secretion. The scrotum was normal. The external meatus was moist and reddened and a mucoid, viscid discharge could easily be expressed. A smear stained with methylene blue was negative for intracellular diplococci but a few nonspecific organisms were present. The fresh discharge was mixed with an equal quantity of physiologic saline solution and a hanging drop examination revealed many pus cells, motile trichomonads and a few red blood and epithelial cells. Rectal examination showed a moderately enlarged and tender prostate with the median groove palpable. The seminal vesicles were not palpated. Microscopic examination of fresh prostatic secretion expressed after the patient had voided revealed 25 to 35 pus cells per high power field with many clumps, a few motile spermatozoa, motile trichomonads and a decreased number of lecithin granules. The urine was cloudy in both glasses and contained many motile trichomonads in a centrifugalized specimen. Local treatment was not given because of the marked balanitis. The patient was instructed to cleanse the penis and keep it clean by the use of warm boric acid soaks. He returned the next day with improvement in the swelling and was able to retract the prepuce with slight difficulty. A posterior instillation of two cc. of 0.25 per cent silver nitrate and a three minute anterior instillation of five cc. of 0.25 per cent silver nitrate were given. The patient returned two days later stating that he felt much better. The nocturia had diminished to four times. The swelling of the prepuce was markedly lessened. There was a slight urethral discharge but examination of the fresh secretion did not reveal motile trichomonads. Reëxamination of the prostatic fluid revealed trichomonads. The urine was much clearer. He was given a three minute anteroposterior injection of a 1:1000 aqueous solution of acriflavine hydrochloride. Two days later he returned to say goodbye, stated that he felt fine and did not wish any further treatment or examination and that he was leaving for the North.

TREATMENT

Davis and Colwell[26] showed by direct toxicity tests under the low power of the microscope that the following substances caused immediate loss of motion in the organisms: glycerine, 50 per cent or stronger; alcohol, 25 per cent or stronger; compound solution of iodine, in dilutions up to 1:100, mercuric chloride, in dilutions up to 1:5000; compound solution cresol up to 1:400; liniment of soft soap, one per cent or stronger; silver nitrate, two per cent or

stronger; and that several other chemicals produced similar effects in a period of a few seconds or a few minutes. In vaginal infestations the treatment recommended by Greenhill[3] is widely used, and Goodall[27] reported uniformly successful results in 22 consecutive cases with the use of cones containing one per cent trinitrophenol. Grimm[23] stated that the treatment of trichomonas urethritis is still in an undecided stage, that injections of mercury oxycyanide 1:4000 often give rapid improvement but that after apparent cure the discharge can recur. Riba[24] has found aqueous acriflavine hydrochloride and weak silver nitrate solutions to be of value and his finding would appear to be borne out by the case recorded here.

COMMENT

The literature pertaining to trichomonas infection of the urinary tract has been reviewed. Trichomonads were found in freshly voided urine by Künstler in 1883; the parasite has been found in bladder urine by several observers; in the case reported by Lewis and Carroll the organism caused a severe acute pyelitis and the same was probably true in the case reported by Visher. Rosenthal has also drawn attention to the possibility of the organism ascending to the renal tissue. At this point it might be of interest to note that in the female genital tract the parasite evidently is limited to the vagina. Holmes[28] reported one patient who had the cervix cauterized nine times without relief from the irritating discharge and another patient who had a panhysterectomy without relief from the discharge, showing that the infection does not necessarily depend on a cervical or uterine lesion.

Twelve cases of a definite urethritis in the male have been reviewed and an additional case added.

The examination of the fresh urethral and prostatic secretions in cases of nonspecific urethritis is emphasized. This is the simplest form of microscopic examination and can be readily carried out wherever there is a microscope. The organisms are more easily detected if the drop of fresh secretion to be examined is mixed with an equal quantity of physiologic saline solution.

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MORTALITY RATES

Telegraphic returns from 85 cities with a total population of thirty-seven million for the week ending March 18, indicate a mortality rate of 12.1 as against a rate of 13.9 for the corresponding week of last year. The highest rate (24.6) appears for Long Beach, Calif., (resulting of course from the recent earthquake), and the lowest (5.5) for Fort Wayne, Ind. The highest infant mortality rate (13.8) appears for Flint, Mich., and the lowest for Akron, O., Duluth, Minn., Somerville, Mass., Tacoma, Wash., and Wilmington, Del., which reported no infant mortality.

The annual rate for 85 cities is 12.4 for the eleven weeks of 1933, as against a rate of 12.5 for the corresponding period of the previous year.

MEDICAL PROGRESS

PROGRESS IN THE STUDY OF CARDIOVASCULAR DISEASE IN 1931

BY SYLVESTER MC GINN, M.D.,* AND HOWARD B. SPRAGUE, M.D.*

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I. Introduction

THE medical literature of 1931 contains many contributions to the cardiovascular system presented by a wide group of workers. Although no investigation has resulted in a discovery of paramount importance, the accumulation of all the findings in the respective fields of the subject have greatly added to our understanding of the cardiovascular system.

Of particular interest, is the progress being made in the investigations of rheumatic fever both as to the significance of general etiological factors and as to the rôle played by the streptococcus. Modifications in the therapy of rheumatic fever may eventually follow the numerous preliminary reports on vaccine therapy.

The hemodynamics generally and of special conditions have been studied, simplifying the very complex changes in which the cardiovascular system takes part. The clinical application of such changes is being utilized in devising various functional tests which are so important to those devoting their time to insurance work.

In the therapy of cardiac conditions, there have been reports on two relatively new methods of treatment, the use of sulphocyanates and the use of glucose given intravenously with insulin. Further reports are essential before the medical profession is warranted in using them routinely.

It is the purpose of this review to summarize not only the more important investigations but also the work that is representative of any certain field, indicating the progress that has been made in the study of cardiovascular disease in the year 1931. It is realized that many worthy contributions have not been included in the bibliography through the necessity of making some limitations. It is hoped, however, that their particular subject has been amply reviewed in the papers selected from that group.

During the year of 1931, several books have been published that are of great value in studying the cardiovascular system. *Bock and Dill* have revised Bainbridge's work on the physiology of muscular exercise. It consists of twelve chapters each one of which treats completely some phase of exercise. *Wenckebach* has published a 120 page monograph in which he presents the modern views concerning the mechanism and hemodynamics of the circulation.

Other monographs are those by *Coburn* on the rheumatic infection and rheumatic heart disease and by *Battro* who has reviewed the physiological and clinical aspects of the T wave of the electrocardiogram.

White has published a volume on heart disease for students and practicing physicians. It is divided into four parts in which there are discussed the methods of cardiovascular examination; causes and types of heart disease; structural cardiovascular defects; and disorders of cardiovascular function. An excellent bibliography has been appended to the text.

The Nelson Press has revised its section on cardiovascular disease in its Loose Leaf Living Medicine. It has included contributions from various authorities such as Maude Abbott who has presented her most recent classification of 1000 cases of congenital heart disease.

II. Anatomy

Patten has described the closing of the foramen ovale as going on in three stages. The development of the pulmonary circulation at the end of embryonic life is sufficient to care for the interchange of gases at birth so that the shunt between auricles may be closed in a gradual manner. At the end of the first postnatal month, the orifice of the foramen ovale is narrowed and the valvula foraminis ovalis undergoes a parallel reduction. For the next six to eight

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months there is a great connective tissue increase of the valvula that may be as much as 70 per cent, and although a probe may still be passed through the orifice, it is now very narrow. Finally, some time during the last third of the first year the valvula becomes adherent to the interatrial wall and is an integral part of the septum. Incomplete adhesion allowing the passage of a probe may be found in about 25 per cent of adult hearts. This is, however, no functional handicap and should be distinguished from the real abnormal conditions where the valvula foraminis ovalis fails adequately to overlap the limbus fossae ovalis.

Stella, by various experiments, rejects Wearn's contention that there is a direct channel from the ventricle via the Thebesian vessels to the coronary arterioles and that the flow is in this direction when the ventricle contracts increasing the intraventricular pressure. He found that the Thebesian vessels are closed to the entrance of blood during systole leaving diastole as the only possible time for such a reversed flow. This, he thinks, is quite unlikely. In normally beating dogs' hearts, there is no evidence of blood passing from the ventricles directly to the coronary arteries regardless of how much lower the pressure is in these vessels than in the cavities.

Grant and Bland report on a study they have made of arteriovenous anastomoses in the human skin and in the feet of hens and ducks. There are many of these anastomoses in the sole of the foot, the palm, and at the tip of the digits. These are the same areas that react strongly to cold, the reaction being most marked where the anastomoses are most numerous. They state reasons that support the belief that these arteriovenous anastomoses have a major rôle in the responsibility for the increased flow of blood in the extremities and should be considered when dealing with the peripheral circulation.

Taussig has recorded some of her histological observations on the heart. She finds that the A-V node is distinctly separated from the auricular musculature but in certain places strands of specialized tissue extend out into the muscle of the auricle. The transition is very gradual so that it is difficult to determine where the specialized tissue ends and the cardiac muscle begins. Contrary to the usual description, she has observed at the lower end of the S-A node, cells resembling those of specialized tissue extending into the auricular musculature. Purkinje cells have been seen extending from the S-A node to the outer wall of the superior vena cava. The specialized tissue cells are larger at the outer margin of the S-A node than are those at the center.

III. Physiology

Writing on dyspnea, *Cullen* and his co-workers found no significant differences either in the arterial or venous blood of cardiac and normal individuals before and after mild exercise when determinations were made of the hydrogen ion concentration, the carbon dioxide content, or carbon dioxide tension. The effects observed were thought to be the result of, rather than the cause of, increased ventilation. They believe the respiratory mechanism is less sensitive to acid-base changes in the blood than is generally conceded and that the cardiac dyspnea is due to some other factor.

The same group of workers in writing about orthopnea believe that this condition is not due to a decreased cerebral blood flow inasmuch as the brain takes the same amount of oxygen in either the sitting or horizontal position. Changes in posture have a negligible effect on the arterial saturation and carbon dioxide content of the blood. The vital capacity, however, is less in the recumbent than in the horizontal position and the change is more marked in patients with congestive failure. This is, they believe, probably the most important factor in the production of orthopnea.

In describing the circulatory adjustments taking place in heart disease, *Weiss* states that dyspnea is one of the earliest signs of cardiac failure although the velocity flow, minute output, and capillary pressures are unaltered. In the earliest stages, the lactic acid and oxygen carrying power of the blood are the same in cardiac patients as in other individuals. Some change, then, must take place in the pulmonary system itself and not necessarily elsewhere. He believes that the reserve capillaries of the lungs are filled up as pulmonary pressure increases in beginning congestive failure, new alveoli are opened up with rigid walls giving a functional emphysema, and decreasing the vital capacity by increasing the residual air. Gravity affects the pulmonary pressure by fostering the sedimentation of the blood in the dependent parts of the lungs and further decreasing the vital capacity. Animal experiments have shown that dyspnea in the early stages is caused by nervous connections between the pulmonary circuit and the medulla, other features of a slowing circulation becoming factors in causing dyspnea later. He also describes right-sided heart failure as a gradual process because blood backs up in the peripheral circuit and so relieves the heart. When the left side fails, however, there is an immediate increase in pulmonary capillary pressure as the left ventricle is unable to expel the blood being sent to it continuously by the efficient right ventricle, and severe dyspnea results.

Means has briefly reviewed dyspnea in a general way and *Levine* differentiates bronchial and cardiac dyspnea by the type of râles, the former being squeaking and musical, the latter, moist.

Jones has also reviewed briefly the physiological processes involved in the production of cyanosis and the changes found in clubbed fingers. This latter condition he states may occur in 35 to 40 per cent of the cases of congenital heart disease, and in 50 per cent of the cases of subacute bacterial endocarditis.

Harris and Lipkin state that the cardiac outputs at work and at rest show the reserve power of the heart; the smaller the difference the lower the reserve. Cardiac output stands inversely to the percentage utilization of oxygen. The increase of stroke volume while at work in relation to the increase in utilization of oxygen, then, indicates the degree of the cardiac reserve. A normal individual responds to work with an increase in stroke volume but a diseased heart responds with an increased pulse frequency.

Moore has recorded the respiratory movements and oxygen absorption curves of the lungs in fourteen experiments on dogs from which data he has calculated the cardiac output and the fractions of blood passing through each lung separately. With free breathing the relative amounts of oxygen absorption in the right and left lungs were in the ratio 3:2. After pulmonary collapse brought about by occlusion of a bronchus, it was found that the circulation to the affected lung was decreased, the cardiac output and circulation to the opposite lung also being decreased but to a lesser extent. He concludes that the diminished circulation in a collapsed lung is the result of both local changes and a decrease in cardiac output.

Fine and Drinker have likewise shown in rabbits that the right lung has a greater blood volume than the left and that both absorption and compression atelectasis reduce the total blood volume in the affected lung.

Another study on cardiac output was made by *Powers* on dogs in which had been produced experimental mitral stenosis. The cardiac output was a normal average of 171 cc. per kilogram a minute in four dogs but much more, 323 cc., in the fifth animal. He also found an elevation of the basal pulse rate, an increase in the red count at the expense of the plasma, and an enlargement of the cardiac shadow by x-ray.

Eyster states that whether or not a high venous pressure leads to cardiac muscle injury with dilatation and hypertrophy seems dependent upon the extent of the venous pressure and the protective influence of the pericardium. Excepting for great excesses, the pericardium is sufficient to prevent injury but when it is absent myocardial damage may result from high venous pressure associated with muscular exertion. It is of importance, then, to protect the heart from high venous pressures, for once dilatation and hypertrophy have been initiated, constant exposure results in changes greater than would otherwise appear. The venous return to the heart, according to *Henderson,* is dependent upon the pressure exerted by the musculature of the body and is the chief factor in determining the volume of the circulation. In illness, there is a loss of muscular tonus and a consequent failure of venous return.

In a short paper, *Blumgart* has discussed the velocity and volume of blood flow. He says that the volume blood flow through the lungs is determined by the "Fick principle" which consists of estimating the total quantity of oxygen absorbed in or carbon dioxide given off from the lungs in a unit time divided by similar estimations made on 1 cc. of blood simultaneously as it passes through the lungs. The velocity of the blood flow through the lungs is determined by finding the time interval between the appearance of an active deposit of radium in the right auricle and its appearance in a large artery of the arm. An increased body metabolism, emotion, exercise, anemia and epinephrin are factors which increase cardiac work and so increase the volume blood flow and the velocity of blood flow. In another paper *Blumgart* and his co-workers presented two cases of coarctation of the aorta. They found that the velocity of blood flow to the legs was reduced but under resting conditions the oxygen supply to the tissues was normal. They were unable to explain the hypertension in the upper extremities of these cases since they observed no differences in the degree of arteriosclerosis of the arms and legs and the arteriolar pressure of the former was normal.

Studies were made on the hemodynamics in two other conditions. *Ellis and Weiss* investigated five cases of complete heart block, two without functional disability, two with moderate and one with pronounced disability. The two without disability were relatively young and had normal blood pressures, the other three having systolic hypertension. Venous pressures were normal in the absence of congestive failure but the metabolic rates were toward the lower limits of normal in all cases. The circulating blood volume was decreased in four patients but the stroke volume for four patients was 40-50 per cent above normal. These findings are thought to be compensatory responses to the slower heart rate.

Smith has made a study of the circulatory changes in a patient with an arteriovenous aneurysm for six years. An operation was performed closing the fistula and it was observed that the cardiac output had been reduced 58 per cent and an increase of 122 per cent in the coefficient of utilization was noted. These findings reverted to something approaching their preoperative figure as the closure broke down and a recommunication was gradually established.

Calhoun and his group of workers have presented several papers concerning the potassium

content of muscle. They found that the muscle of a dog's leg that has been overworked as a consequence of stimulating the sciatic nerve shows a diminution of potassium as compared with the opposite unexercised leg. Further investigation created the belief that overwork causes a loss of potassium from heart muscle and that this loss is one of the predisposing factors to cardiac failure. Because it is known that the potassium content of cardiac muscle of individuals dying of congestive failure is less than in others, a study was made to see if this diminution was due to digitalis. Twenty-seven dogs were used, ten were controls and ten were given "therapeutic" doses of digifolin comparable to that of man. Their cardiac muscles contained a slightly lesser amount of potassium than did the controls. The other dogs were given toxic and fatal doses of digifolin. They showed a distinctly lower potassium content than did the controls. The content of the skeletal muscles of the dogs given digitalis was no lower than the content of the controls.

Scott showed that there is no appreciable difference in the average of the amounts of sodium or potassium in the hearts of edematous and non-edematous patients.

Weinstein and Weiss studied the blood serum in 75 cases of hypertension and in twenty-five control cases with reference to the potassium calcium ratio and the inorganic phosphorus and cholesterol. Slight rises were found in the potassium and cholesterol levels of the hypertensive patients but the calcium level remained normal. They conclude, "Neither changes in the potassium calcium ratio nor hypercholesteremia can be considered as playing a fundamental rôle in the development of arterial hypertension. Elevation of the potassium and cholesterol levels observed in one group of patients with hypertension is the result rather than the cause of changes in the cardiovascular system in arterial hypertension."

Raab presents experimental evidence showing that the symptoms of essential hypertension result from a lack of oxygen and accumulation of lactic acid in the vasomotor centers as a result of local circulatory disturbances due to spasm or sclerosis.

Lambert believes that cardiac pain is the result of stretching the adventitia of the coronary vessels and the aorta due to stimulation of the sympathetic endings there. The former become dilated by an accumulation of blood that the weakened myocardium is unable to propel further. Any sudden rise in blood pressure or encroachment on the nerve endings by inflammatory processes causes dilatation of the aorta and consequent pain. A fall in blood pressure, as by the use of the nitrites, he states allows a relaxation of the blood vessels and a cessation of pain. Another paper on cardiac pain is that by *Sutton*, the experimental work and subsequent discussions showing that cardiac pain in the unanesthetized dog and monkey is caused by ischemia or anoxemia of the heart muscle. The coronaries were completely or partially occluded and the resulting pain as judged by the distress of the animal depended on the degree of closure. It was observed that pricking the parietal pericardium produced pain but pulling did not. Neither did mechanical or chemical irritation of the visceral pericardium, nor of the myocardium provoke a pain response.

Observations on the heart during exercise have been made by several writers. *Boas* found that the heart rate fell so rapidly after exercise that to gain a true estimate one must record the rate continuously during the work. The major drop is within a minute, but the rate is still elevated after an hour.

Vaquez states that in well-compensated cardiac disease there is little or no change in the mean pressure following exertion. In cardiac insufficiency, especially with failure of the right ventricle, the mean pressure becomes elevated on exertion and returns slowly to its resting level.

Bramwell and Ellis examined a group of marathon runners clinically, roentgenologically, and electrocardiographically. This type of running is compared with other distances in relation to oxygen intake, requirement, and debt.

Borman and Meek, working on dogs, have destroyed the sinoauricular node by applying radon to it. No permanent nodal rhythm was established, but a coronary sinus rhythm evidently originates in the nodal tissue of that point. It is suggested that a coronary sinus rhythm cannot be differentiated in the electrocardiogram from a sinoauricular rhythm and that this may be the mechanism in cases where the sinoauricular node is damaged by disease. Cycles of auriculoventricular block were observed and possibly mean interference with the transmission of impulses.

IV. Pathology

Several writers have presented cases of pathology of the pulmonary artery. *Means and Mallory* report a case showing that there may be a total thrombotic occlusion of a main branch of the pulmonary artery without damage to the lung providing the development is gradual. *Boswell and Palmer* had a case with progressive thrombosis of the pulmonary artery with canalization following an upper respiratory infection. Shadows seen in the x-ray were later proved to be thrombi at autopsy. *Costa and Arnett* have separately reported cases of congenital pulmonary stenosis. The former's patient was twenty years old and had coëxisting patency of Botallo's duct, a closed interventricular septum but did have a cribrate condition of the foramen ovale. The latter's case, that was thirty-three years of age, showed no cyanosis

until thirty-one years old and had no other cardiac deformity. *Wahl and Gard* report a case of a pulmonary aneurysm opened by mistake and resulting in fatal hemorrhage. They have added fourteen new cases of pulmonary artery aneurysm proved at autopsy, bringing the total in the literature to 70.

MacCallum reported a case of obliterative pulmonary arteriosclerosis, the only one found in 12,000 autopsies although there have been a few over forty recorded in the literature. *Brenner* believes that atheroma of the intrapulmonary branches of the pulmonary arteries is quite common and that it is sometimes accompanied by intimal thickening in the smaller arterial branches. Among seven cases of pulmonary arterial pathology he has presented, there is one of endarteritis obliterans of the small pulmonary artery branches in a case of mitral stenosis and is probably of rheumatic origin.

Paine and Platt think that pulmonary arteriosclerosis is preceded by a stage of chronic bronchitis with mucopurulent sputum. The cardiac stage follows this, first showing a period of cyanosis and finally right-sided heart failure.

Hein has reported five cases of narrowing of the aorta at its isthmus and also states that the diagnosis of coarctation of the aorta proximal to the ductus arteriosus is almost impossible clinically because of the lack of characteristic symptoms.

Wood believes that the diagnosis of dissecting aneurysm of the aorta is difficult to make but presents a case where it should have been suspected. His patient fell suddenly unconscious while complaining of a severe pain between the shoulder blades. The collapse was very similar to that of a coronary thrombosis but the localization of the pain was unusual.

Schwab and Sanders gave a case of an aortic aneurysm rupturing into the conus arteriosus of the right ventricle. They remark that in one hundred cases of aneurysm autopsied, rupture had occurred in forty-seven per cent.

Lower and Farrell believe that in patients having paroxysmal epigastric pains, gastrointestinal hemorrhages, an epigastric tumor, and negative x-rays of the stomach, an aneurysm of the splenic artery should be considered. Pancreatic involvement may be present. Absolute hemostasis at operation is essential with or without splenectomy for those cases died in which a tampon was employed.

Several unusual cases have been reported. *Bland, Balboni and White* record a tremendous heart, the displacement volume of which was 4600 cc., six or seven times normal, the left auricle itself holding 1760 cc. The patient was a young man with mitral disease who led a relatively active life until the time of his sudden death. A case of deficiency of the pericardium was described by *Beck* who notes that in no instance was this anomaly the cause of death in those cases reported in the literature. *Yater* has given a comprehensive discussion on tumors of the heart and pericardium and has brought out the clinical signs by which they may be suspected or diagnosed.

The pathologic changes in the hearts and the other tissues of rabbits and guinea pigs were studied by *Rake and McEachern* after hyperthyroidism had been produced by intramuscular injection of thyroxine. Similar tissues were studied in a control group and the conclusion was that the differences were quite insignificant. These same workers compared the hearts of twenty-seven patients who died with hyperthyroidism with one hundred and fifty control cases. No myocardial changes were found to account for the cardiac manifestations of thyrotoxicosis.

The results of experimental work in yellow fever have been published by *Lloyd*. The disease was produced in twenty monkeys by injecting the yellow fever virus. Bradycardia was observed in all but one animal, persisting after bilateral section of the vagus, and becoming more marked from day to day. The electrocardiogram showed prolonged auriculoventricular conduction time in eighty-four per cent of the cases, occasional P wave changes, and various alterations in the T wave in seventy-four per cent of the cases. This latter fact is thought to be indicative of myocardial degeneration since the two animals who recovered did not show it. Jaundice and bradycardia are associated inconstantly whereas this is not the case in yellow fever. The histopathological examination of the myocardial tissues of animals who had died of yellow fever revealed well-marked degenerative changes in the musculature of the sinoauricular and auriculoventricular nodes, the auricles and the ventricles. Fatty, granular, hyaline, and vacuolar types of degeneration were seen. These changes are thought to be consistent with the functional changes evidenced by electrocardiography.

A condition of the aortic valve in which there are marked deposits of calcium in the valve cusps causing aortic stenosis has been described independently by *Tuohy and Eckman, Margolis* and his associates, and by *Christian*. It occurs usually in men, is consistent with a long life, causes cardiac enlargement, and the classical symptoms of aortic stenosis. Christian also notes that there is frequently a history of rheumatism early in life and that there may be roentgenographic evidence of calcification in the region of the aortic valve.

Bolton and Barnard in discussing portal stagnation state that when any obstacle hinders the return of blood through the inferior vena cava to the heart, the resulting increase of venous pressure passes back through the liver to the portal vein. Due to the distention of the splanchnic area, a high portal pressure sufficient

to maintain compensation is impossible and the arterial pressure falls. The marked venous stagnation results in necrosis and cellular degeneration; there is an increase in the lymph production; it becomes stagnant and leaks away from the liver as ascites. Later the volume of blood increases and the pressures go up with a resultant increase in blood and lymph flow through the liver. Compensation is partially effected and with the establishment of anastomoses is almost complete. In congestive heart failure, recovery of the heart removes the obstruction and compensation is restored although the liver and great veins may remain permanently dilated. Further cellular necrosis must be considered due to a recurrence of congestion. *Zak,* also, discussing the liver in decompensation, states that in chronic engorgement of the liver in patients with heart disease, there is a disturbance of the normal relation between the liver and water content of the tissues. With congestion of the liver the tissues of the skin become filled with water and thus the venous part of the circulation is unburdened.

Cobb believes that the capillary and arteriolar anastomoses keep the arterial tree from being the sole source of blood supply to any part so that there are probably no end arteries in the true sense. The blood pressure in the small anastomosing vessels is low and the pressure from a surrounding edema may very effectively shut off their blood supply. It is possible in this way in the brain for the occlusion of an artery to cause enough reduction of oxygen supply to result in necrosis. A relative anoxemia is sufficient to cause necrosis of brain cells for they are more dependent on their oxygen supply than any other tissue. It need not be a complete ischemia. Another study on the blood vessels of the brain was made by *Tuthill.* In twenty-four cases of hypertension he found arteriosclerosis of the vessels of the medulla oblongata in only twelve, the remaining cases having very slight or no sclerosis. Six cases with normal pressures showed sclerosis of those vessels. He concluded that the theory of hypertension being the result of arteriosclerosis of the vessels of the medulla oblongata was unsubstantiated.

Larson has reviewed thirty-six cases of mesenteric vascular occlusion in which a necropsy was performed. The most frequent cause of arterial embolism was mural cardiac thrombi; of arterial thrombosis, arteritis and arteriosclerosis; and of mesenteric venous occlusion a septic process of the ascending type. The arteries were affected in fourteen cases, the veins in sixteen, and both veins and arteries in six, these vessels being, in all except one case, of the superior mesenteric group. When intestinal infarction took place, it was generally manifested by symptoms indistinguishable from intestinal obstruction. The course of the disease is short, often less than forty-eight hours, and definite peritonitis is found in about fifty-three per cent of the cases.

Sarasola discusses the etiology and pathogenesis of postoperative embolic thrombosis. He believes that there are three essential factors in the predisposition for the formation of a thrombus. The first is an alteration in the circulation that may result from postoperative debility due to trauma, lack of sleep, loss of blood, and the toxic effect of the anesthetic on the myocardium. The second cause is the alteration in the composition of the blood that may be due to the serobacteriologic or physicochemical alterations observed in the blood of postoperative patients. The third thrombophilic factor is the alteration of the vascular endothelium which may be due to the appearance of noxae which provoke endothelial reactions.

Vance states that there are two types of severe fat embolism each having a different clinical picture with a definite pathological lesion. The first is the pulmonary type occurring about two days after injury in which there is a marked obstruction of the pulmonary arterioles and capillaries, the patient dying an asphyxial death. The second is a cerebral type in which the embolus gets into the systemic circulation and then into the brain. Death in coma may follow in two to seven days as a result of involvement of the central nervous system. *Rukstinat* has found in experiments that dogs whose coronary arteries are plugged with air die promptly. In air embolism, death or recovery may be prompt depending upon the quantity of air. Cerebral irritation succeeds a cerebral air embolus but no case of cerebral air embolism was observed in dogs recovering from coronary artery air embolism.

De Vecchi described inflammatory processes taking place in children affected by various toxic infective diseases but in which subsequent thrombosis fails to appear. He believes these changes are important in understanding the pathogenesis of the endocarditic process and the development of valvular defects. *Moon and Stewart* report an interesting experiment in which they inoculated unsensitized young dogs and rabbits with streptococcus viridans obtained from a man with bacterial endocarditis. The clinical features and both gross and microscopic examinations were those of rheumatic fever. These results strengthen the evidence of rheumatic disease being caused by streptococci of low virulence.

Munck has reported on one hundred and thirty autopsied cases of adults dying a sudden death from heart failure. In seventy-four cases, mainly between the ages of fifty and seventy, death was due to coronary artery disease; in twenty-three cases it was due to myocardial or valvular disorders, while syphilitic aortitis

caused death in thirty-three cases or twenty-six per cent, at an average of forty-five years.

Etiology

A. CONGENITAL

The results of the clinical electrocardiographic and radiologic examinations of one hundred and nineteen children having probable congenital heart disease have been published by *Perry*.

Lichtman adds two new cases of dextrocardia to the literature and reviews the subject of this condition completely.

Sprague, Bland and White report a case of congenital idiopathic hypertrophy of the heart in a seven months old female child. The heart weight was five times that of the normal weight at that age. An interesting family history was obtained in that a sister died of a probably identical condition, a half sister apparently has a patent ductus arteriosus, an aunt has rheumatic heart disease, and a great aunt has an undetermined cardiac disease.

Collins has reported an interesting finding discovered at the autopsy of a sixty-nine year old man. An anomalous papillary muscle was attached to the pulmonary valve of the heart. He reviewed the subject of anomalous chordae tendineae and papillary muscles.

B. RHEUMATIC INFECTION AND RHEUMATIC HEART DISEASE

Swift discusses the factors favoring the onset and continuation of rheumatic fever which he says is one of the most important diseases economically, inasmuch as it is the cause of thirty to forty per cent of the chronic cardiac disease in the latitude of the North Atlantic states. Climatic conditions, poverty, malnutrition and mild toxic states favor its development whereas physiologic stress and strain determine its localization. Most patients give the history of having repeated nonspecific infections over a period of years so that it seems justifiable to consider the infection as having a long course of preparation and sensitization of the tissues before sudden outburst of rheumatic fever occurs. The repeated relapses are also evidence of mild activity persisting until set off by a severe focal infection. It follows then that therapeutic and prophylactic efforts directed against these mild infections may be most beneficial.

In an epidemiological study of fifteen families in New Haven, *Paul and Salinger* show that the spread of rheumatic fever through these families strongly suggests an infectious process with the important factors of heredity and environment yet to be determined. The appearance of rheumatic activity in one individual is often accompanied by manifestations of the disease in other members of the family and not infrequently by other types of illnesses such as bronchitis, bronchopneumonia, or skin rashes.

In another paper, *Paul* writes that contrary to the old belief which had included secondary attacks, the greater number of rheumatic fever patients have their primary attack in the first decade. It is important to note from the viewpoint of transmissibility of the disease that occasionally nursing infants contract the disease from their mothers who are in an active phase. Children between the ages of five and twelve in families in which there is at least one individual who has had rheumatic fever stand a twenty-five to thirty-five per cent chance of acquiring the disease. In this same group, but of individuals who have had their primary attack, the chance for a recurrence was thirty-five and six-tenths per cent. The most susceptible age is between four and twelve years with the peak at seven years.

Longcope states that available statistics indicate that rheumatic fever is rare or unknown in the tropics. At the Johns Hopkins Hospital in Baltimore, in five years, rheumatic fever in all its forms and stages constituted 1.37 per cent of the total medical admissions. Of a series of one hundred and forty-two cases studied during this time only six showed signs and symptoms of arthritis alone, but seventy-seven per cent showed evidence of arthritis during their stay in the hospital. Rheumatic fever apparently differs somewhat in Baltimore as compared with other localities in that severe acute arthritis is rarely seen and in that it is essentially a disease of the heart, preceded or not by mild arthritis or chorea.

Shapiro and his associates (See *Seham*[174]) found that fifty-four per cent of eight hundred and nine children admitted to a heart clinic had no heart disease and that 74% of the remaining children with pathology had rheumatic heart disease. In the cases of early mitral disease it was possible to obtain a history of rheumatism in ninety-two per cent. In addition to the usual clinical tests, he finds that a positive esophagram reveals a dilatation of the left auricle and is positive in fifty-five per cent of the cases of mitral regurgitation, sixty-seven per cent of the cases with double mitral disease, and in seventy-seven per cent of those of double mitral disease and aortic regurgitation.

Several workers have made observations on the association of rheumatoid arthritis and the hemolytic streptococci. One group working with *Cecil* has found almost constant presence of streptococci in the focus of infection; has recovered them from the blood in sixty-two per cent of rheumatoid patients as compared with 3.8 per cent of the controls; and has recovered them from rheumatoid joints in sixty-seven per cent of the cases, but found none in nonrheumatoid joints. They have been able to reproduce rheumatoid arthritis in animals by injecting these organisms. There is a disappearance of agglutinins with recovery from the symptoms

of arthritis. *Nicholls and Stainsby* found a strong specific agglutination reaction with streptococci recovered from the blood and joints of patients. They also noted a close antigenic relationship between "typical strain" streptococci and the hemolytic streptococci from scarlet fever and erysipelas. *Collis* reports an epidemic of tonsillitis caused by the hemolytic streptococcus in a ward of rheumatic children. Rheumatic relapses occurred in nine of eleven children who had the infection but they were unable to isolate the streptococcus from their blood.

Koroboff and Mekler studied 663 cases of various arthritic infections and found the heart to be unharmed in 9 per cent of the acute and 33 1/3 per cent of the subacute articular rheumatic conditions. Endomyocarditis begins in the first days of the development of the articular rheumatism as they found it in 86.6 per cent of the cases. True endocarditis never improved without leaving some kind of a mark. Mitral stenosis is not diagnosable until the sixth month after the infection and usually requires eight to twelve months to develop.

Baudet reports a case of acute articular rheumatism starting out with symptoms very closely resembling appendicitis. An appendectomy was performed and a normal appendix was found. The next day the diagnosis of acute articular rheumatism was made, salicylates were given, and the patient eventually was cured. The patient's condition apparently was not modified by the operation. *Sheldon* states that relapses of acute rheumatic arthritis or carditis are often preceded by a fever one to four weeks previously, most frequently due to a demonstrable upper respiratory infection. The removal of the tonsils, he believes, does not influence the likelihood of a rheumatic relapse.

Slater describes three cases of acute rheumatic fever in which there was closure of the coronary arteries. He explains it as due to the general involvement of the vascular system in acute rheumatic fever. *Thayer* discusses the points of similarity between subacute and chronic vegetative endocarditis and subacute rheumatic heart disease, namely, the distribution most commonly on the mitral and aortic valves and the subacute course. He then describes the various clinical features that aid in the differential diagnosis, but draws attention to the fact that occasionally non-hemolytic streptococci may be recovered from the blood in rheumatic fever and that mural thrombi may form and give rise to peripheral emboli.

Waggoner has presented a good review of Sydenham's chorea in which he states that fifty per cent of his own cases had either cardiac enlargement or evidence of rheumatic fever.

Intracutaneous tests were made by *Derick and Fulton* with the purified nucleoprotein fraction of a hemolytic and green streptococcus on 670 patients. Positive tests were found in 88.1 per cent of the patients suffering from rheumatic fever and in 54.9 per cent of normal individuals. In children under 15 years of age, the proportion of positive to negative tests increases with age. Other illnesses of a streptococcal nature also give high percentages of tests. The hemolytic streptococcus nucleo-protein usually gives stronger reactions than does the green streptococcus antigen. In four cases of subacute bacterial endocarditis only one case showed a positive test and that was to the hemolytic streptococcus nucleoprotein. The test has no specific diagnostic value because positive reactions may be obtained in a variety of diseases.

C. SUBACUTE BACTERIAL ENDOCARDITIS

Gluchov summarizes in part his experiments on forty-four rabbits. He produced septic endocarditis by spraying toxic doses of streptococci on the lymphoid tissue of the throat. Immunization with streptococcus vaccine protected the animal against subsequent toxic doses, but it did not protect against the staphylococcus.

Morse reports on 100 children seen in their first attack of acute endocarditis 10 to 30 years ago and of this group 36 are dead, 3 are cardiac invalids and 61 are alive and well. Thirty-seven of these patients in the latter group have normal hearts. Only one of the living patients has a mitral diastolic murmur. The results depended more upon the care that the children received than upon any other factor. In the cases in which rheumatism seemed to be the cause, the results were not so good as in those in which an infection of the upper respiratory tract was the apparent cause.

Various aspects of subacute bacterial endocarditis have been described by several writers. *West* reports a case of an apparently healthy thirty-one year old woman, who while playing tennis, died suddenly of a coronary occlusion as a result of an embolus dislodged from a bacterial growth on the aortic valve. The resultant discussion revealed other sudden deaths in persons without known heart disease.

A femoral aneurysm was seen by *Craig* during the course of an illness of subacute bacterial endocarditis in childhood. *Doherty and Trubek* describe characteristic elliptic retinal hemorrhages with white centers occurring in the bacterial endocarditides, acute and subacute, and in the severe anemias. They have no prognostic value.

A complete list of references will appear at the end of this article which is to be continued.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 19131

TWO WEEKS' FEVER AND ABDOMINAL PAIN

MEDICAL DEPARTMENT

An Italian housewife thirty-seven years old entered the hospital complaining of abdominal pain.

For four weeks she had had almost continuous dull abdominal pain. For two days before entry she had had pain over the epigastrium on inspiration. Ever since the onset she had had headache and chill, sometimes at night. She had had occasional slight cough. Her bowels had been regular. For two weeks she had been in bed on a diet of milk only.

The past history is negative, the family history not significant so far as obtained.

Physical examination showed a woman with evidence of loss of weight, lying flat in bed breathing rapidly with an occasional expiratory grunt. The lips were dry and cracked, the tongue dry. The skin was dry, with no areas of discoloration. No enlargement of the heart was made out; the stomach tympany encroached on the left, however, so that the apex impulse was not seen or felt. No abnormalities of the heart or lungs were noted. The blood pressure was 95/60. The abdomen was full and tympanitic. There was slight tenderness over the pubes. Several lumps, not tender, were felt in the course of the colon. The liver and spleen were not enlarged. The extremities, pupils and reflexes were normal.

The hemoglobin was 85 per cent, the white cell count 4,000, 10,000, 2,500, the smear normal; no parasites. The urine showed a trace to a slight trace of albumin at three of four examinations, granular casts twice, red blood cells once. The specific gravity was 1.018 to 1.006. The stools showed a positive guaiac on two examinations. A skin tuberculin test was negative. An eye consultant found the fundi normal.

The temperature was 100° to 106°, the pulse 98 to 128, the respiration 23 to 40.

Two days after entry a few faded brownish papules were noted on the abdomen, back and shoulders. Some of them disappeared on pressure; most did not. On the fourth day the patient said she felt well except for dry mouth. There were crackles at the right base behind. The next day the alae nasi moved, there was grunting expiration, squeaks were heard in the left front and harsh breathing at the left base. The patient complained of some abdominal pain. There was moderate distention; no tenderness, spasm, or evidence of fluid. She was very clear mentally. On the sixth day she vomited several times. That night she vomited after taking milk and spat out a mouthful of fresh blood after vomiting. There were many crackles throughout the right chest, front and back. For the next two days she continued to vomit repeatedly, often with an added mouthful of blood which now seemed clearly to come from the gums. The vomitus was clear greenish fluid which gave a positive guaiac and on one occasion showed one ascaris lumbricoides. With small doses of morphia she had no vomiting on the tenth and eleventh days, and retained fluids in considerable amounts. On the thirteenth day she began to vomit again and retained nothing by mouth. Rectal feedings were at once expelled. She failed steadily and on the fifteenth day died.

CLINICAL DISCUSSION

DR. RICHARD C. CABOT: This sounds like an acute infection, temperature, pulse and respirations all looking like it; I do not know where, but very likely above the diaphragm.

Ordinarily the house officer does not put down "expiratory grunt" unless he thinks the patient has pneumonia. I do not know why the point about "discoloration" is put into the record. The blood pressure is notably low, as it often is in pneumonia, tuberculosis and other infectious diseases.

Two of the white cell counts were notably low. Of course with evidence preceding this pointing to an infection one wonders whether these cells were driven down, as they often are, by an overwhelming pneumonic infection.

"The stools showed a positive guaiac on two examinations." So far we have nothing else to link up with that.

"A few faded brownish papules were noted on the abdomen, back and shoulders." Those are not typhoid rose spots if they are properly described. They are not the right color, and the blood is outside the vessels and not inside; but typhoid is what they had in mind.

If this was typhoid—and we have to consider that—we should be surprised if the patient was "very clear mentally".

This is certainly not at all a clear case. This chart shows a continued fever practically never below 102°, a pulse that is between 100 and 120 most of the time and a respiration somewhat elevated. The main point is that it is a steady fever, not touching normal in the two weeks

that she was here. The chart also shows some diarrhea or loose movements, which I did not get before. We are dealing then with a continued fever lasting two weeks, and any diagnosis we make has to be a diagnosis of the causes of that known condition. The other most definite thing about it is this blood which is shown in the stools apparently twice, and comes by vomiting a number of times. Then we have some crackles in the lungs, especially in the right lung. We have a low white count twice out of three times. If they did a Widal they are not telling us what it showed.

Miliary tuberculosis cannot be excluded. It is one of the things which could cause such a fever as this. One hesitates to make that diagnosis when there have been no suggestions of tuberculosis in the past history. There is no such suggestion at all. In a great majority of cases we have some history of a previous process, healed or arrested. Still I do not see how it can be excluded.

Typhoid fever and miliary tuberculosis are the two things which seem to me most probable in this case. The Widal will probably decide between the two. We do not have a Widal in tuberculosis. We almost always do have it in typhoid.

Can it be any form of septicemia, on the heart valves or in any localized abscess process? I do not see that we have anything to suggest this. Is there anything to suggest malignant disease anywhere? I do not see it. Those lumps said to be in the course of the colon of course might conceivably be lymphomata. We can get fever with a lymphoma. We can get a blood count as low as this with some cases of lymphoma. The shortness of the history, however, is the main point against lymphoma,—five weeks to death. That is too short. I do not believe it is that. I believe it is an acute infectious disease, and I do not see what it is unless it is one of the two I have mentioned.

Can it possibly be influenza? What time of year was this? How many years ago was this?

MISS PAINTER: It was in January, 1912.

DR. CABOT: There was no epidemic of influenza in 1912.

A STUDENT: Could the ascaris infection be the cause?

DR. CABOT: The ascaris infection cannot have been of any importance.

A STUDENT: How about ascaris pneumonia?

DR. CABOT: No.

A STUDENT: Ascaris intestinal obstruction?

DR. CABOT: We have no evidence of obstruction. Ascaris can cause obstruction.

A STUDENT: She vomited continually.

DR. CABOT: Not continually, nor was it the characteristic type of vomiting.

A STUDENT: Cannot ascaris go into the lung tissue?

DR. CABOT: No. Nor can it cause fever. It seldom does any harm. I do not believe it caused any harm in this case.

A STUDENT: Liver abscess?

DR. CABOT: I do not see that we have any evidence of it. The signs of liver abscess are enlargement of the liver, chills and pain if it gets to the surface. Many of them of course are entirely latent. With such low blood counts I do not think anybody could ever make the diagnosis. I am against liver abscess.

A STUDENT: Can't you exclude miliary tuberculosis by the negative tuberculin test?

DR. CABOT: I should not think so. We sometimes get negative tuberculin reactions in such cases, but I should say that was neutral in significance here.

A STUDENT: How do you rule out neoplasm?

DR. CABOT: I do not see how you can rule it out, but I cannot rule it in. For such a diagnosis we have to have some disturbance of some function or part, and we cannot localize this in any particular place. I never make a diagnosis of neoplasm unless I can put it in some place. Can it be in the stomach? She is only thirty-seven years old. I never saw fever like this in carcinoma of the stomach. I do not believe it.

A STUDENT: As part of the trouble can she have scurvy, causing bleeding from the gums?

DR. CABOT: Yes, but she has had enough good food here to get her over it.

A STUDENT: Can that be purpura?

DR. CABOT: No, I do not think we can say a person has purpura unless she bleeds from a good many different places. One has to have subcutaneous bleeding.

A STUDENT: How about the brownish papules?

DR. CABOT: I do not think those were any evidence of bleeding.

A STUDENT: What about lung flukes?

DR. CABOT: I never saw a case in my life. I am against it, because I am sure I should have heard of it if we had ever had a case of it in this hospital.

A STUDENT: She had ascaris, so she might have been exposed to other parasites. Why doesn't the ascaris count?

DR. CABOT: Because it is just a harmless partner.

A STUDENT: Ascaris apparently migrates. It can get through the intestinal wall apparently, and would not an infection through there migrate through the diaphragm?

DR. CABOT: All I say here rests on what I have seen in these autopsy findings in the last thirty years.

A STUDENT: According to Cecil's textbook this whole picture can be accounted for by ascaris.

DR. CABOT: I do not find anything like this in Cecil. I do not believe it is that. I think

it is more likely to be typhoid than anything else. Against that we have the absence of any rose spots and the clear state of mind, which is not at all characteristic. We have no positive serological evidence in the blood, but we have no negative either. I do not think I can exclude miliary tuberculosis, but that seems less probable because of the absence of previous evidence of tuberculosis. I should say, therefore, I am uncertain between these two, with a preference for typhoid fever.

BACTERIOLOGIC REPORTS

A blood culture taken on the third day showed typhoid bacillus.

Widal tests on the first, fifth and eleventh days were negative. One on the fourth day was highly suggestive.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Typhoid fever.

DR. RICHARD C. CABOT'S DIAGNOSIS

Typhoid fever.

ANATOMIC DIAGNOSES

1. *Primary disease.*

(Typhoid fever.)
Typhoid ulcers of the ileum and cecum.
Typhoid hyperplasia of the mesenteric lymph glands and the spleen.

2. *Secondary or terminal lesions.*

Hemorrhagic edema of the lungs.
Hydrothorax, left.
Edema of the kidneys.

3. *Historical landmarks.*

Slight chronic pleuritis, right.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: The autopsy showed a perfectly typical typhoid, with ulcers scattered throughout the ileum and a few very minute ulcers of the large intestine. There was nothing else of any importance in the case.

CASE 19132

GRAVE CONSEQUENCES FROM A CHRONIC DISCHARGING EAR

EYE AND EAR INFIRMARY

An American draftsman twenty-seven years old entered complaining of discharge from the right ear for eight years, from the left ear for many years, soreness in the right side of the jaw for six days and deafness for four days.

The discharge from the right ear was intermittent, and had followed a mastoid operation. The discharge from the left ear was more profuse than that from the right. Two months before admission he had a right upper tooth removed. Some local soreness had persisted. Six days before admission soreness in the right side of the jaw near the ear developed, diagnosed by his physician as rheumatism and treated with powders which the doctor told him would cause dizziness and vomiting. He had not been dizzy, but had vomited after each powder for the past three days. For one day he had had vertical headache and postaural tenderness. Since taking the powders he could hardly hear at all. He had been slightly deaf for years. The day before admission and also the morning of admission, in the Outpatient Department, he had a temperature of 102°. He had had no chills, sweats, stiffness of the neck or incoördination of movements.

The family and past history are negative except that he alone in his family had always had a scaly, thickened epidermis.

Physical examination showed this condition. The heart and lungs were normal for either. Local examination showed the right ear-drum red, with a large posterior bulge. A paracentesis was done, with release of considerable pus and blood with great relief. The drum was found to be very thick. The left ear showed a large central perforation of the drum with granulations and slight injection along the handle. The gums were very red and were bleeding in places. The teeth showed much dentistry. Behind the right ear there was swelling which was very tender on pressure. The hearing of both ears was limited to conversation at a shout. Rinné's test was 0/25 on the right, 0/19 on the left. Weber was positive on the right, negative on the left. There was no reaction of vertigo or any other symptom after syringing both ears with 40 cubic centimeters of ice water. The fundi were normal.

The urine was negative. The white blood cell count was 17,000.

Before operation the temperature was 101.5° to 100°, the pulse 80 to 72, the respiration normal.

X-ray examination showed complete sclerosis of the left mastoid. The right mastoid showed a postoperative cavity. There were no cells remaining.

Two days after admission operation was done. Two days later the patient was in satisfactory condition. On the sixth day his temperature rose to 104.5° and he complained a great deal, was apathetic, somewhat irrational and fearful of being hurt. He vomited several times. Later in the evening the surgeon found him unconscious. The pupils reacted to light. There was no spontaneous nystagmus. There was marked stiffness of the neck and a positive Kernig. An eye consultant found no choking of the disks but marked engorgement of the veins of the right fundus. A lumbar puncture gave fluid

of a cloudy, ground-glass appearance under an initial pressure of 325, final pressure 75, hydrodynamics normal. The fluid showed 16,000 cells, 87 per cent polynuclears, 12 per cent lymphocytes, 1 per cent transitionals. Globulin was present, sugar absent. The total protein was 618, the colloidal gold 5555555555. Culture showed no growth. There was insufficient fluid for any other test.

That day a second operation was done. The next day the condition was poor. The neck was very stiff. There was bilateral Kernig, questionable Babinski, no nystagmus. The patient could be awakened but was irrational. Another lumbar puncture that night gave fluid at an initial pressure of 400, final pressure 100; 10,000 cells, 88 per cent polynuclears, 12 per cent lymphocytes; globulin a trace, sugar a trace; culture, no growth. For the next week the condition was fairly good. He was irrational at times and complained of headache. There was considerable drainage from the wound. By the twenty-third day he was doing well, up in a wheel chair with no complaints. There was a large herniation from the wound which slowly became smaller. On the forty-ninth day after the second operation the patient was discharged improved.

Clinical Discussion

Dr. Maurice Fremont-Smith: I think it is worth emphasizing that when we are dealing with a chronic discharging ear we are dealing with a bundle of dynamite. The ear may go on discharging for years and nothing happen; on the other hand you are never safe. The discovery of a discharging ear may throw great light on a difficult diagnosis.

"The discharge was intermittent and had followed a mastoid operation," which means I suppose that there are certain mastoid cells which were not cleared up at that operation. All my remarks are subject to help and correction by Dr. Mysel, whom I see sitting here, because he knows a great deal more about ears than I do.

What do you suppose those powders were? Salicylate or perhaps morphia. We do not expect dizziness from morphia. The danger of making the diagnosis of rheumatism when there is pain perhaps will be illustrated here. Where was this pain? It was in the region of the jaw. Had it to do with that tooth, perhaps osteomyelitis of the jaw, or was it above the jaw, high up in the glenoid fossa, in the region of the mastoid? We do not know as yet.

"He had vomited after each powder for the past three days." It may have been suggestion, but on the other hand he was not dizzy. Perhaps the powders had nothing to do with it, and that would be significant. He may have been vomiting without any other inducement than his disease. They were quite strong powders apparently.

Of course the question of deafness may become very important. The amount of involvement in a middle ear may be indicated by the amount of deafness. You have a child with an earache and a little temperature and a drum that is red and a little bulging but not very much. I believe that one of the important indications of serious trouble, perhaps one of the indications for paracentesis, is deafness in that ear. I have seen an ear man test a very young child by going to the other side of the room and whispering, "Don't you want a piece of candy?" The response was immediate on the good side and delayed or absent on the affected side!

"Posterior bulging" means nothing to me. The bulge was outward or not at all. I suppose it means in the posterior portion of the drum. How could that be described as a "large posterior bulge"?

Dr. Philip Mysel: This "large posterior bulge" referred to a mass of granulations coming through a posterior marginal perforation.

Dr. Fremont-Smith: "Behind the right ear there was a swelling which was very tender on pressure,"—mastoiditis of that side which they had opened. Rinné's test — the tuning fork placed against the mastoid, no sound heard by air conduction and sound heard for 19 seconds by bone—indicates middle ear involvement as against nerve involvement. The Weber test is done by putting the tuning fork on the vertex of the skull. The fact that it was heard on the right indicated that the right eighth nerve was active; it should be heard also on the left, that is if the nerve were intact. Of course the fact that he had middle ear disease in both ears would tend to make the sound heard in both ears. It is much more difficult to interpret a Weber in a patient who has both ears involved. What does a negative Weber on the left mean when the Rinné shows that there is bone conduction on both sides? That is not consistent, is it?

Dr. Mysel: This patient had bilateral middle ear disease. Weber lateralized to the right means that the right ear shows a greater loss of hearing than the left.

Dr. Fremont-Smith: There was no reaction after syringing both ears with 40 cubic centimeters of ice water. Normally there would have been.

Dr. Mysel: Yes. In a normal person you get a reaction after five cubic centimeters of ice water. This test should have been carried on further, up to at least 120 cubic centimeters of ice water, before one could feel that there was no vestibular response. The caloric reaction showed hypofunction of the right labyrinth.

Dr. Fremont-Smith: That would indicate that the disease had involved the labyrinths on both sides.

Dr. Richard C. Cabot: Is that the Bárány test?

DR. MYSEL: Yes; Bárány was the first to describe the caloric test.

DR. FREMONT-SMITH: "X-ray examination showed complete sclerosis of the left mastoid." We do not know whether any mastoid cells were left. We may be able to see them in the x-ray.

DR. MYSEL: The left mastoid had not been operated on.

DR. FREMONT-SMITH: There was sclerosis, which means the effect of long-continued infection on that side. On the right we do not know whether there were any cells. Evidently there was something from which the constant discharge was coming. If the mastoid cells were all removed where was that coming from?

DR. MYSEL: From the tympanic cavity. In this type of middle ear disease you have bony necrosis of the annulus, and this is usually associated with cholesteatoma.

DR. FREMONT-SMITH: And a radical mastoid operation consists in removing those.

DR. MYSEL: He had had a simple mastoid eight years previously.

DR. FREMONT-SMITH: They are going to operate for an acute or subacute mastoid on the left. The problem now arises, what has he more than that? Has the infection gone through the mastoid and given a little localized meningitis? Has he a lateral sinus thrombosis? Has he possibly a brain abscess? Or has he a meningitis? Those are the things in our minds. The fact that he had headache does not necessarily prove any of those more serious conditions, but it raises the question. He has had no chills. We expect chills in a lateral sinus thrombosis. We do not know as yet about his physical examination, whether he had a stiff neck. We do know that his fundi were negative, but one can have an abscess of the brain without evidence in the fundi of increased intracranial pressure, since it takes some time for choking of the fundi to develop. The things we should like to know are whether he did have a stiff neck, whether he had any changes in his reflexes, whether he had any nystagmus, which he apparently did not have, because the labyrinths were hypo-active; whether he had a Kernig; whether his fields showed anything. Going ahead of the game a little and assuming that he may have an abscess, it would probably be located in the region of the temporal lobe, and we know that the fibers of the optic radiations run through and spread out so that if you have a temporal lobe abscess you may get a hemianopsia. That is what we always look for in considering a tumor in that region. From a mastoid one might get an abscess below the tentorium, in which we should get cerebellar signs.

DR. CABOT: Have we the x-rays?

DR. GEORGE W. HOLMES: The dark shadow in the center is the internal auditory canal and the triangle here is the mastoid region. This area of diminished density on the right I presume represents the operative field. There are no shadows on either side that one could interpret as being mastoid cells.

DR. FREMONT-SMITH: He had an old process in that mastoid. We have no further information about the neurological findings before operation. Did you see him before operation?

DR. MYSEL: He had a negative neurological examination before operation.

DR. FREMONT-SMITH: So there is no reason to think of anything except chronic mastoiditis on that side?

DR. MYSEL: Except that his pulse was a little low for his temperature—a pulse of 80 and a temperature of 102°—and he was somewhat drowsy. We rarely see in ear cases a pulse as low as 80 and a temperature of 102°.

DR. FREMONT-SMITH: That makes me wonder whether one ought not to find something more in the way of neurological signs. If we explain his low pulse by increased intracranial pressure, as we are tempted to do, there would probably have been something to go with it in the way of neurological signs.

DR. MYSEL: He had localized headache and temperature, but no stiff neck, no spontaneous nystagmus, and no Kernig; the rest of the neurological examination was negative. The fundi were negative; the fields were not done before the first operation.

DR. FREMONT-SMITH: Won't you tell us about the first operation?

DR. MYSEL: I should like to clear up a few things about the history. The powders that the doctor gave him and that made him quite deaf I am inclined to think were quinine rather than salicylates. It is not unusual to have pain referred to the angle of the jaw in ear cases, due to reflex disturbance from the fifth nerve.

There is quite a difference between chronic ears. We rarely see an intracranial complication in a discharging ear from a central perforation, whereas intracranial complications usually follow marginal types of perforation. When you see a large central perforation associated with obscure neurological symptoms, you are not so much worried as if you see the marginal type of perforation. The latter causes bone necrosis and is associated with infected cholesteatoma, and you may get any kind of intracranial complication such as a brain abscess in the middle or posterior fossa, meningitis, suppurative labyrinthitis or sinus thrombosis, or a combination of these complications.

When I saw this man he had a temperature of 102°, his pulse was 80, he was a little drowsy, and he had this bilateral otitis with central perforation on the left and marginal on the right. He had had some postaural signs, tenderness and pain at the angle of the jaw. All we could say was that he had an acute exacerbation of the chronic otitis on the right. His pulse and

drowsiness made us a little suspicious that something else was going on.

A radical mastoid operation was done. He had had a simple mastoid operation eight years previously. Of course simple mastoid operations have no place in chronic suppurative ears, because the infection is in the middle ear and not in the mastoid. On exposing his tegmen there was found a large patch of infected granulations, in other words a large extradural abscess which covered the middle fossa and extended over the mastoid tegmen to the outer surface of the right temporal bone. This was the right ear and a right-handed man. He had no aphasia. Aphasia may be present sometimes in an extradural abscess of large size when it is on the left side. His dura however was a little tense, and I was a little suspicious that the extradural abscess did not account for the whole picture. The wound was left wide open.

DR. FREMONT-SMITH: You were looking for spontaneous nystagmus in an attempt to place the lesion below the tentorium?

DR. MYSEL: Spontaneous vestibular nystagmus is the chief labyrinthine symptom, and tells us whether the labyrinth has been irritated or invaded by disease. From the symptoms and the type of nystagmus one can usually tell whether the infection has spread through the labyrinths or into the ventricles. Cerebellar abscess usually gives a coarse horizontal nystagmus to the affected side. Meningitis gives a vertical or a mixed type of nystagmus.

DR. FREMONT-SMITH: We have then signs of meningitis. Are there organisms in the spinal fluid, or have we a so-called "aseptic meningitis"—a term which Dr. Cabot rightly criticizes—in which there are signs of meningitis, a high spinal fluid cell count, yet the sugar will be normal because there are no organisms and a culture will be sterile? In this type the prognosis is entirely different from that of a streptococcus or pneumococcus meningitis, in which the prognosis would generally be absolutely bad. In sterile meningitis the patient may have meningeal symptoms and a high cell count for days, but if sepsis does not develop and the drainage is adequate, may entirely recover. So we are much interested to find what the spinal fluid showed.

They probably tested the jugular pressure to find whether he had a sinus thrombosis. If you press on the side on which there is thrombosis you will get no rise and fall, and if there is no sinus thrombosis you will usually obtain rise and fall on jugular compression.

"Globulin was present, sugar absent." Until we reach this point I do not think we can say the prognosis is absolutely bad. I believe that now we must say there are organisms in the spinal fluid and the prognosis is bad. He has meningitis. "The total protein was 618," tremendously high. "The colloidal gold was 5555555555," as we should expect in any type of meningitis. "Culture showed no growth." I think that probably means that there were organisms there but very few, or that for some reason they did not get a good culture, and we are nevertheless dealing with a meningitis.

I assume the second operation was done to produce better drainage, probably opening the dura and if possible finding and localizing an abscess.

DR. MYSEL: Following the first operation his headache rapidly subsided, the pain disappeared, and he was feeling very comfortable. We observed him for a period of six days. During this period his temperature ranged from 100° to 99°, his pulse from 60 to 70, but on two occasions his pulse went as low as 54. Except for his slow pulse there was absolutely no sign of brain abscess. His fields were negative.

I saw him on the morning of the sixth day and he was feeling very well. That night he suddenly became irritable and apathetic, and when I saw him an hour later he was unconscious. He had no spontaneous nystagmus and his pupils reacted to light. That was the only favorable thing in the whole picture. If he had had spontaneous vertical nystagmus with what we felt to be the underlying cause, namely brain abscess, the prognosis would certainly have been hopeless.

His wound had been left open. The brain was explored and a large abscess was evacuated and drained. He finally recovered.

So far as meningitis goes, I do not think there is any question but he had a true meningitis. The mere fact that we do not find organisms in the spinal fluid does not mean that there is not a true meningitis. He had temperature, headache, stiff neck, Kernig, all the classical signs of meningitis. Recently Fraser and Logan Turner reported their cases for the past twenty years, and some of their cases coming to autopsy showed no organisms in the spinal fluid during life but showed a diffuse leptomeningitis at autopsy.

DR. FREMONT-SMITH: I do not see how we can explain the absence of sugar except by the presence of organisms.

DR. MYSEL: I think he probably had some organisms there.

DR. FREMONT-SMITH: Is it not extremely unusual to get organisms in the spinal fluid and have recovery?

DR. MYSEL: We have had several cases recover after one positive culture, but rarely after a second or third. Personally I believe that you can have organisms in the internal auditory meatus without having organisms in the spinal fluid. One does not usually expect to find organisms in the blood stream following an abscess of the arm.

DR. FREMONT-SMITH: The only thing that seems consistent in this is the fact that he

had no sugar. If he had had any sugar I should think that he might recover; finding no sugar I should expect him to die.

A STUDENT: Do you have to take the cells into consideration in the absence of sugar? Don't they say there are two things that use the sugar, bacteria and cells?

DR. MYSEL: He had 16,000 cells.

DR. FREMONT-SMITH: That may explain it.

A STUDENT: Was a definite diagnosis of brain abscess made before the second operation?

DR. MYSEL: Yes; that was the one thing that we suspected. With an extradural abscess and this sort of picture it usually means brain abscess, and not meningitis without brain abscess.

DR. FREMONT-SMITH: Was it in the temporal region?

DR. MYSEL: In the right temporal lobe. If it had been in the left he would probably have had aphasia and that would have helped us in localizing it at the first operation; but without that to go on and with the extradural abscess, he had enough to account for his symptoms.

A STUDENT: Didn't he have remarkably few symptoms?

DR. MYSEL: You can have a brain abscess in a latent stage without any symptoms. It may be present for weeks, months, even a year, then suddenly break into the ventricle and give rise to meningitis and death in a short time.

A STUDENT: Do you suspect that he had that before he entered the hospital?

DR. MYSEL: There is no question in my mind but that he had the abscess before he came in. It was a chronic type of abscess.

DR. MAURICE FREMONT-SMITH'S DIAGNOSES

Mastoiditis.
Meningitis.

DIAGNOSES (FROM HOSPITAL RECORD)

Chronic otitis media, right, with mastoiditis.
Right temporosphenoidal abscess.

The New England
Journal of Medicine

SUCCESSOR TO
THE BOSTON MEDICAL AND SURGICAL JOURNAL

Established in 1828

Published by THE MASSACHUSETTS MEDICAL SOCIETY under the jurisdiction of the

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SUBSCRIPTION TERMS: *$6.00 per year in advance, postage paid for the United States, Canada $7.04 per year, $8.52 per year for all foreign countries belonging to the Postal Union.*

Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the Journal office, 8 The Fenway.

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Communications should be addressed to The New England Journal of Medicine, 8 The Fenway, Boston, Mass.

FOREIGN DUMPING ON MASSACHUSETTS

THERE is developing in medical education a movement which may prove to be a serious menace to the health of the people of Massachusetts. American students are flocking to European universities to find the education which they cannot get in the United States. One is reminded of the relatively small group of American students who fifty or more years ago went to Europe, thirsting for the new knowledge and methods that were transforming medical science and practice.

It is interesting to contrast and compare these two movements and note why the present one is fraught with danger and why the former custom of travel and study abroad proved so helpful to this country. At that time the chief centers of medical science and art were in foreign universities and hospitals, and it was necessary for the student who sought proficiency beyond his fellows to find the masters in the older institutions of learning in Europe. The returning stream of enthusiastic teachers and students gave stimulus and direction to a movement in university development in which the present high level reached in many of our schools is but a transition stage to still greater attainments. Now there are centers of medical education in this country which compare favorably with the best in the world: certain of our centers are in some respects without rivals elsewhere.

Why then should American students flock in such numbers to European medical schools, and in what does the menace of such migration lie? It is a phenomenon worthy of closest attention so that its real significance shall be detected and made clear to all.

There is in the United States a demand for medical education far beyond the capacity of medical schools to supply. The schools cannot accept the candidates who desire to pursue the study of medicine. It has been shown that the number of technically eligible applicants for admission to our medical schools each year is approximately thirteen thousand. The number of applicants actually admitted is about six thousand.

Just what becomes of all the rejected candidates no one knows. Some of them appear as the applicants for the following years. Doubtless some give up the hope of becoming physicians, but others migrate to foreign medical schools and a recent estimate is that there are probably two thousand students from this rejected group who are studying in European medical schools and who plan to return to the United States for practice. Study at a foreign university was once a distinction. Grave doubts are entertained as to its value in fitting a candidate for the practice of medicine in this country now. The systems of education are different.

There are grounds for fearing that the standards abroad may be lowered for "foreigners" who are able to pay the tuition fees. Some of the foreign institutions are desperately in need of money, and it is reported that some are willing to grant degrees to candidates to whom the privilege of licensure for practice will not be granted. By this discrimination they protect themselves against possibly unqualified practitioners, but by the same token they meet the requirement of a degree in medicine which has by our laws a commercial value. Such foreign trained physicians will have been without the advantage of clinical work and training which are given to students in our better schools and among them may be many who have been rejected by the better medical schools in this country.

If fears concerning the lowering of standards abroad are realized, every state in the union except Massachusetts can protect itself against

these unqualified practitioners by declaring such low-standard schools to be "not approved". Already state boards of registration with discretionary power of approval, and that means all except Massachusetts, have declared that students matriculating after the school year 1932-33 in foreign medical schools, must have had premedical education equivalent to the generally accepted requirements for admission to approved medical schools in this country, must graduate from a school giving a four year course, and must have successfully passed the examination to obtain the license to practice medicine in the country in which the medical school from which they are graduated is located, if they are to be considered eligible for examination in those states.

This will turn toward Massachusetts, which is unable to protect itself except by examination under the present law, a considerable portion of a distinctly unqualified group of practitioners.

It would be interesting but perhaps idle, to speculate on the possible effect on the "unapproved" schools in this country, the graduates of which can be licensed in Massachusetts only, if this horde of unqualified practitioners is admitted to examination in this Commonwealth. The tooth of competition may prove more penetrating than will be comfortable. It may well threaten their existence. But in any case the public is bound to suffer until the legislature wakens to a sense of its responsibility and protects the people of the Commonwealth by legislation that is not antiquated but meets the situation as it exists.

CONTROL OF SPECIALIZATION IN MEDICINE

SPECIALIZATION in medicine is of comparatively recent origin, and the control of specialization still more recent. In the United States the earliest national society of specialists, the ophthalmological society, was founded in 1864, and the second, that of the otologists, in 1866. No other special national association was created until 1875. In the last fifty-five years many other societies composed of specialists have been formed, and today there is an organization of practitioners interested in every special field of medicine. These associations naturally have a very high standard of membership and require the fulfillment of certain requirements in training, experience, research, and activity in their respective fields before admitting a physician to membership. They are all purely voluntary organizations, with membership attained by invitation and election rather than by application. The membership as a rule is limited and cannot begin to include all men properly qualified to practice the particular specialty.

The need has become apparent for recognizing properly qualified individuals, by creating organizations or boards to which a specializing physician, such as a surgeon or an ophthalmologist can apply and receive a tangible token of his proficiency, if his qualifications are adequate. As a result of this demand, possibly on the part of physicians themselves, possibly by reason of pressure from medical schools and hospitals, or possibly in response to some lay pressure different methods of recognizing ability in a given field have arisen. In the first group are the "Colleges" which confer the title of Fellow on properly qualified members. The American College of Surgeons was established in 1912 for this purpose. Those physicians who passed an examination, including the presentation of definite case reports operated on by that individual, were issued a diploma entitling them to be called Fellows, and the initials F.A.C.S. were conferred as an indication of proficiency. Other "Colleges" have been established since that time.

Secondly, certain groups of specialists have formed boards for the certification of specialists in their particular field. No identifying letters are conferred, but these boards issue certificates and publish, from time to time, lists of accredited specialists who have conformed with the requirements and examinations which these boards have developed. Physicians who receive such certificates are not members of any organization in the true sense, but have been certified as proficient by a board organized for that purpose. The American Board of Ophthalmic Examinations was established in 1916, the Otolaryngological Board in 1924, the Board of Obstetrics and Gynecology in 1930, and the Board of Dermatology and Syphilology in 1932. Several other organizations have taken preliminary steps in the formation of similar boards—the proctologists, the anesthetists, the allergists, etc.

The entrance of a state medical society into the field of certifying specialists of every type has created a third method. New Jersey has recently organized a state committee on specialists with subsidiary county committees for the same purpose. A physician desiring to be considered a specialist applies to the county committee, and if approved, the application is referred to the state committee for final action. Membership in national special societies and adequate experience in a limited field are considered to give the individual a favorable recommendation. Certain safeguards are provided and a fee for certification is required.

If other states take up a similar custom, numerous opportunities for confusion will result. A physician might be a specialist in one state and not in another, and conceivably hold a certificate from a national board or be a Fellow in a "College" and yet not be an accredited specialist in any state, etc. Considerable dupli-

cation of effort is certain to occur unless a coördinating agency is provided. The job of determining the competency of specialists is a national function rather than a state function and the success of the National Board of Medical Examiners in licensing graduates of medical schools argues for a greater coördination of effort in the case of specialization.

It would seem wise, therefore, for the state associations to avoid entering this field until further experience has demonstrated that state control or state supervision is a better method than by a national group. The logical method would seem to be by an organization of representatives of the various special groups which would act in coördinating the functions of the various boards in a comprehensive manner. Each board would be free to deal with the details within its own specialty while the representative body would formulate general policies concerning standards, examinations, etc., and postgraduate training of specialists, at the same time maintaining a close coöperation with the National Board of Medical Examiners and the Council of Medical Education of the American Medical Association, as well as with the various special national societies.

THE USE OF EPHEDRINE AND OF GLYCINE IN THE TREATMENT OF MYASTHENIA GRAVIS

WHILE trying various remedies for myasthenia gravis, a disease from which she suffered, Harriet Edgeworth, Ph.D., a medical student, noted marked improvement after taking ephedrine, grains 1½ in two daily doses*. After two days of treatment she could turn herself over in bed, a movement she had been unable to perform for three years. By experimenting with dosage she found that the best results were maintained on ¾ of a grain daily, an amount which she has taken for the last three years with uniform improvement. "From a totally helpless person dependent on nursing care day and night," she writes, "with difficulty in chewing, speaking, swallowing and breathing . . . I now work about the house, sew, read, write at will, and can get into and out of a car with a little help. The shortness of breath, cyanosis, and persistent insomnia have almost disappeared. . . . Periods of intense heat are very prostrating to myasthenia gravis patients, in fact may be the cause of death. For three summers I have definitely protected myself against this depressing effect by increasing the daily dose during a period of very hot weather.†" These and many other observations made by Dr. Edgeworth are not chance records, but the results of careful research over a period of many months. She is experimenting with the drug in order to avoid any possible adverse effects from long-continued usage. Further reports from such a conscientious observer will be of great use to the medical profession. Already, it would seem, she has made a discovery of considerable importance in the treatment of one of the so-called "incurable" diseases.

A second form of treatment for myasthenia gravis has also been recently advocated. Glycine, an expensive drug, has been administered to five patients at the Mayo Clinic with striking relief of symptoms‡. The dosage given was 10 to 30 Gm. daily, divided into two or three doses. It may be combined with ephedrine.

These two reports, based on a disease which is not by any means rare, although often misdiagnosed, are encouraging and should lead to further trials on a larger group of patients.

*Jour. Am. Med. Asso. 94:1136, 1930.

†Proc. Staff Meetings Mayo Clinic 7:737, 1932.
‡*Ibid.*

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

TRUESDALE, P. E. M.D. Harvard University Medical School 1898. F.A.C.S. Surgeon, to the Truesdale Clinic, Fall River, Massachusetts. His subject is "Low Pericardiotomy for Acute Suppurative Pericarditis. Report of Two Cases and Twenty-four New Cases from the Literature." Page 671. Address: 151 Rock Street, Fall River, Mass.

EINHORN, MOSES. M.D. University and Bellevue Hospital Medical College 1922. Formerly serving in the Gastroenterological Department Mt. Sinai Hospital. His subject is "The Rôle of Psychic Load in Recurrent Attacks of Gastroduodenal Ulcers." Page 681. Address: 983 Park Avenue, New York, New York.

PARVEY, BENJAMIN. M.D. Baltimore Medical School, University of Maryland 1907, and Post Graduate Study in Obstetrics, Gynecology, and Abdominal Surgery, universities of Vienna, Berlin and Budapest, 1928-1929. Formerly, on the Gynecological Service, Boston Dispensary. His subject is "Report of a Case of Acute Hydramnion Treated by Abdominal Puncture." Page 683. Address: 636 Beacon Street, Boston.

SULLIVAN, ELIZABETH ANN. M.D. Tufts College Medical School 1914. Her subject is "Arsphenamine in the Treatment of Vincent's Angina." Page 686. Address: 383 Broadway, Cambridge, Mass.

JENKINS, PAUL K. B.S., M.D. St. Louis University School of Medicine 1924. On the Staff of St. Francis Hospital, Miami Beach, Florida. His subject is "Urethritis Due to Trichomonas." Page 687. Address: Miami Beach, Florida.

MCGINN, SYLVESTER. A.B., M.D. Harvard University Medical School 1929. Dalton Fellow, Cardiac Clinics and Laboratory, Massachusetts

General Hospital. Address: Massachusetts General Hospital, Boston. Associated with him is

SPRAGUE, HOWARD B. A.B., M.D. Harvard University Medical School 1922. Assistant Physician, Massachusetts General Hospital. Visiting Physician and Research Director, House of the Good Samaritan. Assistant in Medicine, Harvard Medical School, Courses for Graduates. Address: 270 Commonwealth Avenue, Boston. Their subject is "Progress in the Study of Cardiovascular Disease in 1931." Page 691.

BOSTON MEDICAL LIBRARY

FOURTH MEETING OF THE BOSTON MEDICAL HISTORY CLUB, FEBRUARY 20, 1933

THE fourth meeting of the Boston Medical History Club was held February 20, 1933. Dr. W. P. Coues presented a brief communication giving Ford Madox Ford's reminiscences of experiences he had at certain mineral baths in England and on the Continent. Others have more successfully dealt with amusing situations arising in the course of their stay in institutions, while undergoing treatment, when endeavoring to point out the foibles of attending physicians in their efforts to apply a particular therapy to the needs of functional neurotics.

The second paper was by Dr. Henry Viets who gave a short review of the life of "James Thacher: Progressive Pioneer Physician of Plymouth." James Thacher was a great man; one of the great figures in American Medicine standing out in a group of a dozen or more revolutionary and pre-revolutionary characters. At 14 or 15 he was apprenticed for five years to Dr. Hersey of Barnstable, who was the physician of his time in that locality. He must have secured a good medical education by the apprentice system for in 1775 when he was twenty-one, he came to Boston to join the Army and along with sixteen others took examinations for surgeon's mate. Many of these were pupils of Joseph Warren, but Thacher secured one of the ten places. He stayed in the Army until 1783, a total of seven years. He never missed an opportunity to meet people and recorded his experiences in a diary. His is one of the most important descriptions of the Revolution; his account of the execution of Major André is the best, extant. After the war when he was thirty-two, he went back to Plymouth to practice and became the leading man in that region. He took on apprentices, often having as many as ten to twelve at one time. He had a school of his own in which he held all the chairs and was a university in himself. Harvard and Dartmouth gave him honorary degrees. He became the best known physician in his County.

He began to write in his middle fifties and published his first book in 1810. It may be that he took to writing because he was becoming deaf, which interfered with his practice.

His first book was a Dispensatory. It was well done, well edited and was well received. He must have had a library, or the beginning of one, as early as 1810. He published nine books that passed through twenty-two editions in the latter part of his life. His next important book was "Practice of Medicine", 1st ed. 1817. This was probably demanded by his students. It was very carefully done and well written. Without a good library, he could scarcely have accomplished this. It was the first good textbook of medicine in the United States. He wrote an excellent monograph on Hydrophobia in which he quoted the English authorities. He also published the American Orchardist. In Plymouth he had an excellent garden, as did many a doctor of those days. Forty years after the War, he published the first edition of the Military Journal, the notes for which were evidently made during the War. They may have been expanded somewhat but he actually kept a diary. This has very little to say about medicine, as Thacher was apparently more interested in the general situation than in medicine. He made out of it a very readable book, for Thacher wrote very well. After his death in 1844, many editions of the Military Journal were published, nine in all.

In 1828 came his American Medical Biography. This has been the foundation of all American Medical Biography. It has not the lightness of touch noted in his earlier works but he was 74 when he began it. He wrote a book on bees, which is the rarest of his works. He wrote his book on Demonology basing it on Salem's experiences with witches. Finally, he published a History of Plymouth to which he added a second edition when he was ninety. His literary activity was enormous. Most of his works were distinct contributions to medicine.

Though he was greatly respected, there were no really good obituary notices at the time of his death. His best portrait is in the Pilgrim Hall at Plymouth, painted by Frothingham when Thacher was about ninety. The portrait in the Boston Medical Library is a copy of that picture. The Library has some manuscripts of Thacher's and a sketch of his life by his daughter. He died at the age of ninety, in 1844.

The third paper was by Dr. Arthur B. Lyon entitled "Some Notes on the History of Infant Feeding." Dating from early Egyptian history, where Isis is represented as suckling the infant Osiris, he traced the change in custom due to the introduction of foreign ideas in consequence of which the wet nurse became the vogue, until by the Ptolemaic period few Hellenized ladies nursed their children. In Babylonia the goddess, Ishtar, is represented as nursing an infant. In India the wet nurse was common but must needs be of the same caste as the parents. In Greece, in Homeric days, mothers nursed their own children. Later on

Athenian women gave over the nursing to a slave, preferably a Spartan. The Jews seem to have been the only race to have adhered to the natural method. In the second to the fourth century an effort was made to put wet-nursing on a standardized plan and Oribasius gives directions as to what should be the qualifications of a wet nurse and also tests as to the quality of her milk. Instead of milk sugar which is, of course, a modern substance almost invariably given during the first few days of an infant's life, the ancients gave honey. Little is said by Greeks or Romans about animal milks but nursing bottles were in use by the Romans in the fourth century. The first pediatric text was printed in 1473 by Bartholomeus Mettinger and was known as "Regiment der junger Kinder." Gruel and milk are recommended until the teeth are cut. Nursing was expected to continue for two years supplemented with gruels and sometimes other foods. De Vallembert, in 1565, was the author of the most common sense book on Pediatrics which had so far appeared. He was quite liberal as to what he gave his babies in addition to breast milk during the latter part of the eighteen months or two years during which he advised breast feeding. In the seventeenth and eighteenth centuries in England infant feeding was far worse than it was in France in the sixteenth. Feeding bottles were unknown in England in Tudor times and nursing was not infrequently carried on for three years. It was in the late seventeenth century that hand feeding began in England and it was well along in the century before any infant welfare movement had really started. High death rates prevailed everywhere. A half of the babies born succumbed to poor feeding. Then came modifications of the nursing bottle—a perforated cow's horn being first used by the English. Experimentation with various kinds of nipples resulted in procuring a fairly good milk bottle. As England became a more prosperous country history repeated itself and the mothers in well-to-do families gave up nursing their infants. A shortening of the nursing period was brought about and cows' milk began to win some favor. Impurities in this type of infant food at first received little consideration but eventually other kinds of food had to be substituted and various sorts of "Pap", combinations of bread and water, sweetened, were tried. Utensils used in feeding were crude and uncleanly. In Georgian England, according to Sir Hans Sloane, the infant mortality was three times as great among dry-nursed as among the breast fed. Wet nursing, in the eighteenth century fell in repute, and attention was turned to perfecting the utensils from which cows' milk could be given. In 1797 Dr. Michael Underwood published in London his "Treatise on the Diseases of Children" and in this work the most useful chapters were on infant feeding. He advocated breast feeding whenever possible but first urged cows' milk and asses' milk with certain modification with barley water. In the nineteenth century came acceptance of the principles of artificial feeding, the production of special foods and the perfection of the nursing bottle. About 1840 the first proprietary infant foods were brought on the market. Until the bringing of adequate supplies of milk into the cities under hygienic conditions it was the custom to drive cows into the city, feed them upon "mash" from the breweries and use city tramp-labor to milk them, with results that might be anticipated.

Though "the world has been made safer for babies" the upshot of it all is that Dame Nature's method is still the best.

MASSACHUSETTS LEGISLATIVE NOTES

S. 352 and **S. 353** have been introduced as emergency legislation to correct the difficulty, recently discovered, which prevents the Board of Registration in Medicine accepting graduates in medicine from medical schools which do not give a full four years' course of instruction of not less than thirty-six weeks in each year.

The text of **S. 352** is as follows:

As affecting applications filed during the current year only with the board of registration in medicine under section two of chapter one hundred and twelve of the General Laws, as appearing in the Tercentenary edition thereof, the words "a full four years' course of instruction of not less than thirty-six weeks in each year", as appearing in said section, shall be taken to mean a course of instruction of more than thirty-five weeks in each year, whether or not inclusive of periods covered by holidays and vacations.

The text of **S. 353** is as follows:

Section two of chapter one hundred and twelve of the General Laws, Tercentenary edition, is hereby amended by striking out, in the eleventh line, the word "thirty-six", and inserting the word:—thirty-two,—so that the second sentence will read as follows:—Each applicant, who shall furnish the board with satisfactory proof that he is twenty-one or over and of good moral character, that he possesses the educational qualifications required for graduation from a public high school, and that he has received the degree of doctor of medicine, or its equivalent, either from a legally chartered medical school having the power to confer degrees in medicine, which gives a full four years' course of instruction of not less than thirty-two weeks in each year, or from any legally chartered medical school having such power, if such applicant was, on March tenth, nineteen hundred and seventeen, a matriculant thereof, shall, upon payment of twenty-five dollars, be examined, and if found qualified

by the board, be registered as a qualified physician and entitled to a certificate in testimony thereof, signed by the chairman and secretary.

EDITORIAL NOTE: The objection to **S. 352** is that it would relieve the situation for the current year only and the objection to **S. 353** is that it would lower the formal course of study by diminishing the required time without giving any guarantee that the quality of medical education would be improved.

NEW HAMPSHIRE NOTES

NEW MEMBERS

Dr. R. A. Hernandez, Laconia, graduated Indiana University School of Medicine 1920, licensed Indiana 1920, New Hampshire 1932.

Dr. Laura G. Jacques, Tilton, graduated Boston University School of Medicine 1930, licensed 1932.

REMOVAL

Dr. Raymond J. Turley, Berlin, to Meredith.

MISCELLANY

A RADIO MESSAGE PREPARED AND SPONSORED BY THE COMMITTEE ON PUBLIC EDUCATION OF THE MASSACHUSETTS MEDICAL SOCIETY FOR THE DEPARTMENT OF PUBLIC HEALTH

THE HEALTH EXAMINATION*

BY ALBERT A. HORNOR, M.D.

A health examination consists of three parts: the story of the case, examination of the body, and laboratory study.

Of these three, the most important part is the story. This story will make the health examination worth while only if the story is told without hurry or anxiety. It should be planned for both by the one who desires the examination and by the one consulted. By this I mean that the medical adviser listening to the story must have plenty of time, and the person undergoing the health examination must know that the doctor, preferably the family physician, has set aside a time for the examination.

First of all, one should tell all his fears and worries.

Secondly, one must tell of any symptoms, however trivial.

Thirdly, one must tell of any change in weight.

Fourthly, one must tell the examiner of any changes one has noticed in one's body.

When you go to your medical adviser for a health examination, remember to tell all of the worries you may have. Be sure to include not only worries about whether you have cancer or tuberculosis or are going crazy, but also worries about whether the house is going to burn up or whether the banks are going to fail. Only if the medical adviser and the patient deal squarely as regards the story, will the health examination give its full value.

All symptoms experienced should be related. Examination of the body and laboratory study will be quite different according to the symptoms described. For instance, a person who complains of dizziness and a person whose feet hurt will need quite a different examination and laboratory study. Be sure to tell all your symptoms to the medical adviser.

Every mother watches the weight of her infant and is proud to tell her friends of the way the baby gains, and this is evidence of good health. So, too, throughout childhood and adolescence one should not only grow but gain in weight. Any failure of a child to grow or gain in weight during a year demands investigation: first of all, of its habits of eating and secondly, a search for any disease that might be present. When one has reached adult life one does not normally lose weight except through design. If you lose as much as ten pounds in weight, report that fact to your medical adviser. Most people gain weight with age. Many of the diseases of old age are dependent upon excess in weight, so that it is important to avoid such gains. Surely if anyone above the age of thirty-five has gained ten pounds in a year, he should report that fact on the occasion of the health examination. The changes which occur in one's body from time to time are often unimportant, e.g., a loss of hair is usually unimportant, but a marked loss of hair should surely be brought to the attention of the physician during the course of a health examination. On the other hand, the appearance of a lump underneath one's skin is a very slight change, but this ought surely to be brought to the attention of the one conducting the examination. The appearance of a lump in the skin, particularly a lump in a woman's breast should lead to an immediate health examination. There are other conditions which are indications for a prompt investigation; in this class belong one who coughs or vomits blood and one who notices a change in his habits of digestion or a change in disposition. So many diseases are mild and curable when they begin that one should have a health examination whenever one notices an abnormal change in one's body or habits.

In addition to the need for health examination upon the appearance of these changes, it has been proved that a health examination once or twice a year is worth many times the expenditure of time and money by the person to be examined. During infancy the health examination should be at least once in three months. Between the ages of two and twenty years the examination should be at least twice a year. Between the ages of 20 and 40 one should have a health examination once a year, and after forty twice a year.

The examination of the body should be thorough.

*Station WBZ, November 15, 1932, 4:15 P.M.

I will not attempt to describe it in detail beyond saying that it cannot be thorough unless the patient is completely undressed. The laboratory study will vary greatly with the age of the person to be examined. The story told to the physician will indicate what laboratory studies need to be made, and the one most needed may be omitted unless the story told is complete and frank.

Get a check-up of your condition once or twice a year, according to your age.

Follow the advice of your medical examiner between examinations.

If any change in your body occurs have a health examination promptly.

If you are worried about your health have a health examination. The facts will surely be better than the suspense.

For many years insurance companies have offered free medical examinations to certain groups of policyholders, and from time to time, have studied the results of hundreds of thousands of these examinations. On each occasion there has been found an important decrease in the death rate of the examined policyholders, as compared with the deathrate of policyholders who did not accept the offer. This must mean an improvement in health as well.

The value of an annual health examination is the same to the body as an inventory is to a business. The health examination shows the condition of the body at the time of examination. Of course people get ill despite having had health examinations, and every doctor is familiar with the fact that predictions of good health should not be made. On the other hand ill health will often be avoided by the remedy of slight defects which have been found on the health examination.

Remember that the most important part of a health examination is the story.

Remember to tell the story frankly to the medical adviser in whom you have confidence.

Remember that a health examination is most often best conducted by the family physician.

FELLOWSHIPS, SCHOLARSHIPS AND APPOINTMENTS AWARDED BY THE HARVARD MEDICAL SCHOOL

Moseley Traveling Fellowships, for study in Europe, have been awarded for 1933-34 to:

Neil Louis Crone (A.B. Grinnell College 1925; A.B. Oxford 1927; A.M. Oxford 1929; Ph.D. Oxford 1929; M.D. Harvard Medical School 1931).

Stanley John Gregory Nowak, Instructor in Surgery, Harvard Medical School (A.B. Harvard College 1923; M.D. Harvard Medical School 1926).

Charles Victor Seastone, Jr., Research Fellow in Bacteriology and Immunology, Harvard Medical School, and Fellow of the National Research Council (A.B. University of Wisconsin 1927; M.D. Harvard Medical School 1931).

The following fellowships have been awarded for 1933-34 to undergraduate students in the Harvard Medical School:

James Jackson Cabot Fellowship to Fiorindo A. Simeone.

James Jackson Cabot Fellowship to Benjamin Alexander.

George Cheyne Shattuck Memorial Fellowship to Harry L. Kozol.

Charles Eliot Ware Memorial Fellowship to Frank R. Pierce.

John Ware Memorial Fellowship to Robert A. Stewart.

The following scholarships have been awarded for 1932-33 to undergraduate students in the Harvard Medical School:

George Haven Scholarship to Henry S. Bennett.

George Haven Scholarship to Ralph E. Durkee.

George Haven Scholarship to William McD. Hammon.

George Haven Scholarship to Richard G. Hodges.

George Haven Scholarship to Milton Landowne.

George Haven Scholarship to William H. Mathews.

George Haven Scholarship to Liberino Patricelli.

George Haven Scholarship to Carey M. Peters.

George Haven Scholarship to Richard B. Pippitt.

George Haven Scholarship to Walter H. Pritchard.

George Haven Scholarship to Waldron M. Sennott.

George Haven Scholarship to Irving D. Townsend.

David Williams Cheever Scholarship to Joseph F. Ross.

Charlotte Greene Scholarship to Edward W. Oxnard.

James Ewing Mears Scholarship to Robert B. Lawson.

The following new appointments have been made in the Medical School:

For one year from September 1, 1932:

Anni Dagmar Seppänen, M.D., Research Fellow in Medicine.

From December 1, 1932 to September 1, 1933:

Karl Erik Fritz Ask-Upmark, M.D., Research Fellow in Neuropathology.

From January 1, 1933 to September 1, 1933:

Frederic Andrews Gibbs, M.D., Research Fellow in Neuropathology.

Philip Mysel, M.D., Assistant in Otology.

John Berell Sears, M.D., Assistant in Surgery.

Gustaf Elmer Lindskog, M.D., Research Fellow in Surgery.

For one year from January 1, 1933:

Leroy Edward Parkins, M.D., Secretary of the Medical Courses for Graduates under the Commonwealth Fund.

From February 1, 1933 to September 1, 1933:

John Arthur Alvarez, M.D., Research Fellow in Medicine.

The following resignation has been received, effective January 1, 1933:

Chapman Hunter Binford, M.D., as Research Fellow in Preventive Medicine and Hygiene.

THE EXHIBIT ON SLEEP AND FATIGUE

Donald A. Laird, director of the psychological laboratory of Colgate University, has been appointed to supervise the exhibit on sleep and fatigue for the Century of Progress Exposition, opening at Chicago in June. The exhibit, which is being supported by the Master Bedding Makers of America, will feature the actual use of apparatus used on recent studies of sleep and fatigue at several universities, giving visitors an opportunity to take some of the tests. It is expected to accumulate in this way a mass of data bearing on the diurnal course of fatigue.

CORRESPONDENCE

AN APPEAL FOR COÖPERATION

City of Boston
Health Department
City Hall Annex

March 23, 1933.

To the Members of the Medical Profession of Greater Boston:

Appreciating in its fullest measure the significant value and importance of greater participation by the medical profession in the practice of preventive medicine, the following routine has been adopted in connection with the program of the Child Hygiene Division of the Boston Health Department under the direction of Dr. Charles F. Wilinsky:

It has been the practice for a number of years for the nurses of the Department of Health to visit new-borns and on this visit the nurse is expected to stress the need of adequate medical supervision of the baby and the preschool child. In every instance in the future, wherever there is a family physician, the nurse will make every effort to interest the mother or guardian to bring the child to his office. In addition to this, she will fill out a postal card containing the following information which will be addressed and mailed to the attending physician:

Dear Doctor:

A nurse of the Department of Health has visited today Baby ....., of ..... Street, ... Suburb. She was informed that you are the family physician and has stressed the advantages of bringing the baby to you periodically for physical supervision and other preventive services.

If the Health Department can be of any further service in getting this baby to your office, kindly communicate with the Child Hygiene Division, City Hall Annex, (telephone Lafayette 5100, line 141).

FRANCIS X. MAHONEY, M.D.
Health Commissioner,
CHARLES F. WILINSKY, M.D.
Deputy Health Commissioner in charge of Child Hygiene Division.

I seek the coöperation of every member of the medical profession in the furtherance of this plan. I am mindful of the fact that their assistance is going to result in a greater appreciation by the laity of the possibilities for the prevention of disease among the young and a realization of the willingness and ability of the medical profession to practice preventive medicine.

Very truly yours,

FRANCIS X. MAHONEY, M.D.,
Commissioner, Board of Health.

COMMENTS ON HEARINGS AT THE STATE HOUSE

March 17, 1933.

Editor of The *New England Journal of Medicine,*

Of late we have read many editorials in this worthy medical magazine about bill No. 106. This bill called on the law makers of this Commonwealth to raise the medical standard in this state. It came up for a hearing before the learned solons of Massachusetts. The Secretary of the Board of Registration in Medicine started to present his bill. Before he was able to utter the first sentence he was interrupted and reinterrupted. Before he started his second sentence another solon asked one of those insulting questions which did not deserve any answer.

There were about four medical men who tried to convince those gentlemen on the committee that bill No. 106 is *just, fair* and *decent.* However, not one of the speakers was given a fair hearing without any interruption. Here is one sample of the questions asked of one of the speakers, "Who sent you up here?" Apparently those gentlemen on the committee could not understand how any normal human being could form an independent idea on any question without the influence of something or somebody.

I was the last one who attempted to defend bill No. 106. I was just finishing a few words when I heard one of the committee complaining that his stomach was empty, and he warned me to finish my remarks before I started. One thing is certain that he realized that I was in favor of the bill which may have made him hungry. In order to save that hungry legislator, I was forced to close abruptly and this was the end of the bill for those who spoke in its favor, and those gentlemen went to have their lunch. Even a child of a primary school could see that that body had made up its mind before coming to the State House on the

morning of February 14. We were informed that one of the members on the committee was the Dean of the school especially interested in the fate of the bill.

A few days after the hearing a banquet was given by the same institution of learning when five members of the above committee attended the celebration. (I wonder whether those members were invited after or before the hearing.) Can anybody be so stupid or innocent as to consider that bill No. 106 had a just and a fair hearing?

While this hearing was going on the student body of that school was well represented.

On March 2, 1933, I was on trial, for I had the opportunity to propose bill No. 1011 which required a physical examination from every applicant for a marriage license. This bill had a hearing before the Committee on Legal Affairs. I received the same courtesy on March 2 as the Secretary of the Board of Registration in Medicine received on February 14. Some of these gentlemen on Beacon Hill do not appear to know that there are such words in the English Dictionary as *courtesy* and *politeness*.

The chairman of the above committee accused me of trying to build up some business for the medical profession. All of them seemed to think that the bill was just a joke.

While I was trying to convince those law makers of the importance of prophylactic medicine, which they did not appear to understand, the newsboys outside tried to prove my arguments.

Now I wish to ask every reader including the Editor, why did bill No. 106 and bill No. 1011 land peacefully in the wastebasket? There were only about five men including one layman to speak in favor of bill No. 106 on February 14. I would like to know where the rest of the medical men who were in favor of this bill were. Did they forget that they are living in a free country where every citizen is entitled to express his or her opinion in the hall of legislation? Do they not know that the solons on Beacon Hill respect, above everything else, Votes?

Where were the physicians who believe and preach to their patients preventive medicine when bill No. 1011 came up for a hearing on March 2? Did they agree with the gentlemen on the committee that the bill was a joke? Where was the Board of Health when that bill came up for a hearing? Even our worthy Editor did not find space in this magazine to mention this bill or the time posted for a hearing. Such an unimportant bill;—a physical examination before obtaining a marriage license, indeed does not deserve even a few lines in a medical weekly in 1933.

Is it any wonder that the vaccination bill has been thrown into the wastebasket for the last 20 years? Is it any wonder that bill No. 106 requiring a higher medical standard for this State was defeated? Is it any wonder that any prophylactic measure has no more chance to pass any committee than anyone of the committeemen has of passing through the eye of a needle?

Let every medical man in this State know the names of those solons who believe in a low standard of medical education and who look on preventive medicine as a huge joke.

I call upon the Editor of this magazine, upon the Board of Registration in Medicine, upon every physician who has respect for his medical degree to throw away foolish prejudice. The enemy is not outside but within the ranks of the profession, which spells "Indifference" and "Let George do it."

Unite for the sake of preventive and scientific medicine.

WILLIAM FRANKMAN, M.D.

RECENT DEATHS

SWEENEY — EDWARD JOSEPH SWEENEY, M.D., of Springfield, Mass., died in that city March 14, 1933.

Dr. Sweeney, a native of Springfield, was born in 1878 and graduated from the Jefferson Medical College of Philadelphia in 1907.

He was a former president of the Staff of Mercy Hospital. He leaves a widow, Mrs. Helen (Foley) Sweeney; two brothers, Dr. Joseph P. Sweeney and Dr. Frank A. Sweeney; and two sisters, Mrs. Catherine Chisholm and Mrs. Mary Kelleher.

GUILD — EDGAR HUNT GUILD, M.D., of Springfield, Mass., died in that city, March 22, 1933. He was born in Ware, Mass., in 1852*, where he began practice after graduating from the University of Pennsylvania School of Medicine, in 1882, moving to Springfield twelve years later. He retired from active practice when he became a member of the Springfield Board of Health in 1908 and was recognized as a bacteriologist of note.

He served as physician to this board until two years ago and during the earlier years of this membership was also clerk. He is credited with a large influence in bringing the work of this department and its hospital to high standards of efficiency. Dr. Guild was a Fellow of the Massachusetts Medical Society and an ex-president of the Hampden District Medical Society. He was a man of high ideals and was fortunate in having a large circle of loyal friends.

He is survived by a son, Lawrence Guild, his wife having died about three months ago and a son a year ago.

*Record as appearing in the Directory of the American Medical Association.

OBITUARY

RESOLUTIONS ON THE DEATH OF DR. HARRY FINKELSTEIN

Whereas, the staff of the Greater Boston Bikur Cholim Hospital has suffered the loss of an earnest and devoted member of its staff in the sudden and untimely death of Dr. Harry Finkelstein,

And whereas, the entire staff of the hospital deeply regrets the loss of Dr. Harry Finkelstein, the first member of the staff to be taken from its midst since the opening of the hospital,

Be it resolved that the staff of the Greater Boston Bikur Cholim Hospital expresses to his family its deep feeling of grief and sense of personal loss at his death.

Be it further resolved that the members of the staff acknowledge his great interest in the patients of the hospital, his earnest efforts in behalf of the hospital, and his untiring devotion and loyalty at great personal sacrifice of his time and health.

Be it further resolved that these resolutions be spread upon the records of the Greater Boston Bikur Cholim Hospital and a copy be sent to his family and also to the *New England Journal of Medicine*.

MAURICE GERSTEIN, *Chairman of Staff*,
NATHAN M. FRIEDMAN, *Secretary of Staff*.

REPORTS AND NOTICES OF MEETINGS

WILLIAM HARVEY SOCIETY

The March meeting of the William Harvey Society of the Tufts College Medical School was held on Friday evening, March 10, at 8 P.M., in the auditorium of the Beth Israel Hospital. Dr. Harry Linenthal, professor of clinical medicine at Tufts College Medical School, presided.

The speaker of the evening was Dr. Burrill B. Crohn, president of the American Gastro-Enterological Association. His subject was "Abdominal Pain." Preceding the discussion of the clinical aspects of abdominal pain, Dr. Crohn presented an excellent summary of our present views regarding the mechanism of visceral pain and its development. The surgeon of the days before anesthesia, was the first to observe the lack of sensitiveness in the viscera to stimuli other than stretching. The sensitivity of the parietal peritoneum, however, was early recognized and Lennander formulated a theory of peritoneal sensitivity in which he stated that pain originates in the peritoneal wall and its subserous layers which are supplied by cerebrospinal nerves. He believed also that the peritoneal lining of the mesenteries was just as sensitive as the parietal peritoneum. Pain would thus be produced by contact between an inflamed part of the abdominal contents and the sensitive peritoneum, or by irritation of the nerves in the root of the mesentery by some inflammatory process.

Ross, however, denied peritoneal sensitivity. He postulated two kinds of pain in disease of the internal organs: (1) splanchnic pain, which is felt in the organ giving rise to the afferent stimuli, and (2) somatic pain, which is felt in the areas supplied by the cerebrospinal nerves to the corresponding segment. Mackenzie further emphasized the spinal mechanism and added the concept of the production of irritable areas in the spinal cord as the result of disease of the internal organs. The theories advanced by Ross were again substantiated by the work of Head, who added the important concept of zones of pain sensitiveness. Disease of the internal organs would thus be characterized by "Head Zones" of pain sensitiveness on the abdominal wall. The methods used in the determination of these zones are well known.

More recently, Hurst, in London, has advanced a theory which is being met with more and more credence. He believes that true visceral pain is merely a question of the appropriate stimulus. Increased tension, such as occurs from the exaggerated peristalsis in intestinal colic, is an example of such an appropriate stimulus. The pain here is not referred but localized. The tenderness and muscular rigidity associated with inflammatory lesions are due to viscerosensory and visceromotor reflexes.

Dr. Crohn then discussed abdominal pain and its significance in the different decades of life. Abdominal pain is rather infrequent during the first ten years of life. The abdominal diseases of children of school age are comparatively few in number. After ruling out appendicitis, the majority of cases of abdominal pain in children can be considered functional in nature. Many cases are closely related to the child's dislike for the schoolroom or the school teacher. Dietary maladjustment should also be kept in mind. A diet too rich in carbohydrates may lead to fermentation within the intestine and pain from the resulting distention. Hurrying at meals may also lead to abdominal pain.

The physician meets with more problems in the second decade of life. Appendicitis should always be borne in mind. Dr. Crohn emphasized the fact that cases of appendicitis may occur in which there is little or no fever. He also denied the existence of "chronic appendicitis" as a clinical entity. Chronic recurrent appendicitis does occur, however, not infrequently. In taking a history, one should endeavor to have the patient describe his pain in his own words. A mere discomfort, fullness, or burning sensation is almost certainly functional. A crampy or lancinating pain, however, is of much more significance.

The differential diagnosis of appendicitis in this age group is often difficult. Right lower quadrant pain in girls at the onset of the catamenia is sometimes due to a ruptured Graafian follicle. The symptoms are frequently indistinguishable from those of appendicitis. There may even be a polynucleosis with a temperature of 101-102°. Ureteral calculus on the right side may give a history identical with that of appendicitis. The patient may show rigidity, reflex vomiting, and polynucleosis. A centrifuged specimen of urine may show no red cells. Cholelithiasis may give similar difficulties.

Terminal ileitis has recently been described by

Dr. Crohn and confirmed by various other observers as a disease entity. The lesion affects the terminal 10-12 inches of the ileum and stops abruptly at the ileocecal valve. The characteristic symptoms are diarrhea and fever. A mass is invariably palpated in the right iliac region at some time during the course of the disease. Sigmoidoscopy and barium enema are negative, but blood and mucus are found in the stools. The lesion is a benign non-specific granuloma causing stenosis and, ultimately, complete obstruction of the intestinal lumen. The reported cases of primary intestinal tuberculosis may very well have been cases of terminal ileitis. Abscess and non-specific granuloma may be the result of foreign bodies in the intestine or of any injury or disease of the mesentery.

The important lesions producing abdominal pain in the third decade of life are gastric and duodenal ulcer. A history of pain coming on at night is of the greatest significance. The pain of perforated peptic ulcer is one of the most severe pains to which man is subject. It is frequently referred to the back and shoulder from irritation of the diaphragmatic peritoneum which derives its sensory supply from the phrenic nerve. Dr. Crohn gave a brief discussion of peptic ulcer in the jejunum following gastroenterostomy. It is important to bear in mind that the pain from jejunal involvement is located rather low in the abdomen. In fact, the only symptom of gastrojejunal ulcer may be pain experienced in the left testicle.

A most important factor in evaluating the symptoms of peptic ulcer is the pain sensitiveness of the particular individual in question. Dr. Crohn described three groups of patients: the normal, the subsensitive, and the supersensitive. The normal person with peptic ulcer will experience abdominal pain during the period of ulcer activity, but will be comparatively free of symptoms while the ulcer is in the dormant state. The subsensitive individual, however, may have no symptoms except during periods of markedly increased ulcer activity, and the supersensitive individual may complain of pain even while the ulcer is in a resting state. In the so-called cases of "silent ulcer," it is really the patient that is silent and not the ulcer. The first complaint of the subsensitive individual may therefore be symptoms of hemorrhage or perforation.

It is interesting to note that tobacco smoke in sensitive individuals may produce symptoms almost identical with those of peptic ulcer. The typical abdominal pain and heart burn are readily simulated and are explained on the basis of pyloro- and cardiospasm.

During the fourth decade, the important lesions to bear in mind in abdominal pain are those of gall bladder disease and pancreatitis. The latter is a fulminating disease and is a frequent complication of the former. One should always bear in mind that the gall bladder case with mild symptoms is ever subject to acute pancreatitis.

The important disease of the fifth decade is cancer. It is ordinarily a painless disease and gives no symptoms unless it involves the inlet or outlet of a hollow viscus or unless there is impingement of the growth on nerves. A typical carcinomatous cachexia with 20% hemoglobin may occur in the complete absence of any pain. Metastases may likewise be quite painless. The younger the individual the lower the level of neoplasms of the alimentary tract. The average age for carcinoma of the esophagus is thus 60 to 65, while lesions lower down the tract are more common in younger age groups. Depressing diseases, including the hormonal disturbances of the menopause, are common in this decade. The symptoms of ulcer and gall bladder disease are often simulated by coronary thrombosis. Young men are no longer excluded from this disease, Dr. Crohn mentioning one case which occurred at the age of twenty-eight.

PLYMOUTH DISTRICT MEDICAL SOCIETY

The March meeting of the Plymouth District Medical Society was held at the Massachusetts State Hospital at the Bridgewater State Farm, March 16.

The meeting was called to order at 11:15 A.M., by the President of the Society, Dr. Charles G. Miles. The records of the last meeting were read and approved. The President of the Society gave a short address regarding House Bill 106, a bill introduced by the Massachusetts Board of Registration in Medicine at the State House to raise the standards of medical education and thereby the practice of medicine in Massachusetts. Resolutions will be brought up later indorsing the State Board of Registration in Medicine in the efforts to improve conditions in Massachusetts. Dr. Miles then read a letter addressed to Dr. George A. Moore, Secretary of the Society, from Dr. Henry M. Landesman asking the Society to support House Bill 519 which reads, "An Act relative to the Liens of Physicians, Nurses and Hospitals for Services rendered in the Treatment of Injuries for which the Patient receives Compensation." The Society voted to indorse this bill.

A nominating committee consisting of Dr. John P. Shaw, Chairman, Dr. Richard B. Rand of Abington and Dr. John J. McNamara of Brockton was appointed and ordered to bring in a list of officers for the ensuing year. The program of the day followed.

The first speaker of the morning was Dr. Burton E. Hamilton of Boston who gave a very instructive talk, taking for his subject "Interesting Points of Angina Pectoris and Coronary Occlusion." He was followed by Dr. Greene FitzHugh of Boston, who had a large number of specimens and demonstrated them while giving a valuable contribution to the interest in the meeting.

A rising vote of thanks was given to the speakers for their very interesting program. President Miles announced at the conclusion of the program that the Society had received a cordial invitation from Dr. Leon Alley, a member of the Society, and Superintendent of the Lakeville Sanatorium, to hold its next meeting on April 20 at his institution. The invitation was accepted with thanks.

The Society then repaired to the large dining hall where an excellent turkey dinner was served. Dr. Wm. T. Hanson, Dr. John Weller and Dr. Maxfield entertained the visiting physicians. A rising vote of thanks was given to the officers of the State Institution for their fine hospitality and entertainment.

The Plymouth District Medical Society wishes to publicly thank Superintendent Sherman of the State Farm for this splendid entertainment and dinner.

JOINT MEETING OF THE GREATER BOSTON MEDICAL SOCIETY AND THE BETH ISRAEL HOSPITAL

The joint meeting of the Greater Boston Medical Society and the Beth Israel Hospital was held on Tuesday, March 7, at 8:15 P.M., at the Beth Israel Hospital. Dr. William Dameshek presided. In the absence of Dr L. H. Newburgh, Dr. T. M. Carpenter of the Carnegie Nutrition Laboratory, Boston, read and discussed Dr. Newburgh's paper on "The Importance of Dealing Quantitatively with Water in the Study of Disease."

Dr. Carpenter prefaced the reading of this paper by certain general remarks regarding water metabolism in the body. He stated that every person in the basal state loses weight at the rate of 0.023 to 0.966 grams per minute through the insensible loss of water. The only time that we gain weight is when we take in food and water. As soon as this is over, the lungs and skin begin the excretion of water, which produces a constant diminution in body weight.

Benedict and Root have shown that the insensible loss of weight is directly proportional to the basal metabolic rate. This was discovered by determining the loss of weight in diabetics who were underweight, and who had a low B.M.R., and also by making similar observations in patients with hyperthyroidism. From these studies, charts were constructed from which one can calculate the basal metabolic rate once the insensible loss of body weight is known.

The experiments that were conducted in making these calculations were fraught with possibilities of error. One patient went out for a short time and returned with some silver change which she had received for a dollar bill. Another removed his false teeth during the experiment, and in both of these instances the change in the weight of the patient was sufficient to introduce a very considerable error into the calculations.

Dr. Carpenter spoke of the ingenious manner in which Dr. Newburgh has utilized the previous information concerning insensible water loss and has added to this store of scientific knowledge. He mentioned particularly the determination of the heat utilized in the evaporation of insensible water loss as constituting about 24 per cent of the total heat eliminated by the body.

Following these remarks, Dr. Carpenter read the paper prepared by Dr. Newburgh and illustrated it with lantern slides. The katabolism of carbohydrate, fat and protein in the body leads to the formation of water; also the breakdown of tissues leads to the liberation of water; these sources coupled with the exogenous water constitute the total supply to the body. Although there may be slight variations from day to day, the water thus supplied to the body strikes a rather constant balance with water given off. In disease, however, this balance is disturbed.

Of especial interest is the water metabolism in adiposity. It was formerly thought that there were two types of adiposity, namely, the simple and the constitutional. Through the work of Dr. Newburgh it has been found that this is a single disease following the law that whatever is added to the body weight must first be ingested. If an obese individual is changed from a diet of 2,300 to 1,500 calories, he loses weight the first two days and then remains at the same weight level. A normal man gained weight even though he was much underfed. These results, paradoxical as they may seem, are the result of water retention.

The studies of water metabolism in nephritis are valuable in considering the treatment of this condition. In both nephritis with edema and nephritis without edema, the ability of the kidney to concentrate waste products is much reduced. It therefore requires an increased amount of water for the complete elimination of the blood elements in dilute form. In nephritis with edema, excretion is difficult, because the water is held so firmly by the tissues that it cannot be utilized by the kidney. In nephritis without edema this added problem is not important. In both conditions, however, forcing of fluids is indicated in order that the kidney may have ample water with which to carry on its function. In nephritis with edema, the subcutaneous fluid does not tend to increase under such therapy, providing salt intake is restricted.

Two case histories were cited, one with edema, the other without edema, in which there was a retention of waste products as evidenced by the high non-protein nitrogen content of the blood. By forcing of fluids the figures were much reduced.

In the discussion of this paper, Dr. Dorothy Gilligan stated that there is a good analogy for the relationship of the insensible water, the water of the vomitus and feces, to that utilized in the pro-

duction of urine. The kidney takes the water that is left over from the other body functions and utilizes it as best it can for elimination of waste products, just as the CO_2 level in the blood is entirely dependent upon the varying levels of the chlorides and organic acid radicals.

Dr. Gilligan continued by saying that there is a definite limit to the concentrating power of the kidney. At 0.3 cc. of urine per minute just 50 per cent as much urea is given off as at 2 cc. per minute. The available fluid for adequate urine excretion should be 2000 cc. daily.

Dr. S. A. Levine considered some of the clinical applications of the studies in water metabolism. In coronary disease, the patient frequently perspires a great deal, and becomes so dehydrated that anuria results. It would be excellent therapy to force fluids in such cases. In cases with rapid respiration, the insensible water loss is increased, and here also the forcing of fluids would be of value.

Dr. Mark Falcon-Lesses, who has worked with Dr. Newburgh, told of how the work on water metabolism was begun. Obese patients were fed on a known diet for some time, and then were weighed each morning. Some gained and others did not, and it was felt that the patients gaining were cheating in their diet. A guard was hired to watch these patients during 24 hours of the day, and still some patients did not lose weight on the undernutrition diets. The only factor that could be responsible for the weight gain was water, and this interesting phase of the body metabolism was studied.

Dr. Herrman Blumgart emphasized the necessity of not restricting water in cases with edema. In cases of nephritis with edema, when one restricts the fluid intake, the patient may suddenly have a diuresis, which is chiefly a water diuresis, and as a result the blood increases in its content of waste products and the patient may develop uremia. Therefore forcing of fluids in this condition is of great importance. An analogy of this phenomenon is the digitalis intoxication in cardiac patients which frequently follows an active diuresis.

MASSACHUSETTS GENERAL HOSPITAL

Fracture Meeting

There will be a conference under the auspices of the Fracture Service Friday evening, March 31, at 8:00 o'clock, in the Lower Out-Patient Amphitheatre.

SUBJECTS

Fractures of the Spine, Dr. W. A. Rogers.
Fractures of the Shoulder Girdle, Dr. Reggio.
Carpal Injuries, Dr. Cave.

House officers, students and visitors welcome.

Philip D. Wilson, M.D.,
Associate Chief, Fracture Service.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

March 30—Clinics in Medicine at Peter Bent Brigham Hospital. See page 665, issue of March 23.

March 31—Massachusetts General Hospital. Fracture meeting. See notice elsewhere on this page.

April 1 and June 13—The American Board of Obstetrics and Gynecology. See page 607, issue of March 16.

April 5—New England Heart Association. See page 571, issue of March 9.

April 12—The New England Dermatological Society. See page 612, issue of March 16.

April 12—New England Physical Therapy Society. Date changed from April 19 to April 12. Detailed notice later.

April 14—William Harvey Society. See page 765, issue of October 27.

May 8, 9, and 10—American Gynecological Society. See page 516, issue of March 2.

October 9-12—American Public Health Association. See page 403, issue of February 16.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

May 3—Wednesday, Annual Meeting. Program to be announced.

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

Tentative Program

Wednesday, April 5, 1933—Essex Sanatorium, Middleton. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Wilson G. Smillie, Professor of Public Health Administration, School of Public Health, Harvard University. Subject: Common Colds.

Thursday, May 4, 1933—Censors' Meeting, Salem Hospital, 3:30 P.M.

Tuesday, May 9, 1933—Annual Meeting. Salem Country Club, Forest Street, Peabody. Golf in afternoon; greens fee $2.00. Dinner at 7 P.M. Speaker: Dr. George Burgess Magrath. Subject to be announced later. Ladies invited.

R. E. STONE, M.D., Secretary.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meeting will be held at the Weldon in Greenfield on the second Tuesday of May.

CHARLES MOLINE, M.D., Secretary.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

May, at the Meadowbrook Club, Reading.

ALLAN R. CUNNINGHAM, M.D., Secretary.

NORFOLK DISTRICT MEDICAL SOCIETY

May—Annual Meeting. Time, place and subject to be announced.

FRANK S. CRUICKSHANK, M.D., Secretary.
1695 Beacon Street, Brookline.

SUFFOLK DISTRICT MEDICAL SOCIETY

Wednesday at 8:15 P.M.

April 26, 1933—Annual Meeting. Election of Officers. Scientific Program—Titles and speakers to be announced later.

The Medical Profession is cordially invited to attend this meeting.

WILLIAM H. ROBEY, M.D., President,
LELAND S. McKITTRICK, M.D., Secretary

WORCESTER DISTRICT MEDICAL SOCIETY

Wednesday, April 12—Dinner and scientific meeting at the Rutland State Hospital. Scientific program to be announced later.

Wednesday, May 10—The Annual Meeting. Place and program to be announced later.

ERWIN C. MILLER, M.D., Secretary.

BOOK REVIEWS

The Science of Signs and Symptoms. By Robert John Stewart McDowall. D. Appleton & Company. Pp. 440. Price $7.00.

This volume is an attempt to "bring clinical work and the sciences closer together". It is in a sense an exposition of applied physiology, and is thus largely a condensation of important physiological knowledge related to disease. It is not an explana-

tion of the mechanism of particular signs and symptoms as the title would imply.

Although there are innumerable facts of great value and interest to be found therein, the reviewer cannot whole-heartedly recommend this book. It is not of practical value in that the physiological data are too complicated for those "who have long since forgotten their sciences"; and the clinical medicine is frequently ridiculously childish. The author is inclined to philosophize and at times is verbose; but he has nevertheless included passages of fundamental facts of great value.

The book seems uneven and would be greatly benefited by covering a less wide field in a more consistently practical style.

Hospitals and Child Health: Hospitals and Dispensaries, Convalescent Care, Medical Social Service. White House Conference on Child Health and Protection. New York and London: The Century Company, 1932. xvii + 279 pages. Price $2.50.

This volume, one of an important series that has come out of the White House Conference on Child Health and Protection, covers the subjects of hospitals, convalescent care and medical social service. In it many facts can be found in regard to the number of hospitals in this country, their situation, bed capacity, and the system of keeping records. A second part of the volume deals with the sources available for convalescent care of children, and a third part, which is perhaps the most important, gives a complete summary of medical social service as it has been inaugurated in hospitals and dispensaries throughout the country. For those who wish to find facts in regard to these subjects this volume can be highly recommended as an authentic reference book. The last chapter, on medical social service, is by Miss Ida M. Cannon, Chief of Social Service, Massachusetts General Hospital, and is of particular interest to physicians of New England.

Textbook of Medicine. By various authors. Edited by J. J. Conybeare. Pp. 1004. Messrs. William Wood & Company, Baltimore. Price $7.00.

The object of this text, written by English physicians, is to offer "the essentials of medicine within as small a compass and at as low a price as possible". To this end there is little discussion of the details of laboratory tests, of the methods of physical diagnosis, or of the technique of special procedures. The book is not a synopsis, but is an admirable brief exposition of the fundamental facts of medicine.

The reviewer feels that the general plan of the book is excellent, with unusually good distribution of topics. The sections on Tropical Diseases and Heart Disease are to be particularly commended. With the exception of the very minor objection to the use of a few terms unfamiliar to American readers, no valid criticism can be made. The remarkable common sense and practical viewpoint render the volume a very valuable addition to any medical library.

Épreuves Biologiques dans Les Ictères. By FRANCOIS THIÉBAUT. Masson et Cie, Editeurs, Paris, 1932. Price 28 fr.

This volume on the biological tests of use in jaundice is written much along the same lines as a previous volume—"Les Fonctions Hepato-Biliares" by the author and M. Chiray in 1930.

This more recent book is divided into four sections. The first deals with the various accepted tests used to study the disturbances of the bile pigments, bile salts, and cholesterin in catarrhal jaundice; toxic and infectious jaundice; Hanot's disease; jaundice due to gall stones; and jaundice due to neoplasm.

The second part deals with the investigation of the internal functions of the liver in which the author includes carbohydrate, water and nitrogen metabolism. The part of the section which deals with the carbohydrate metabolism seems to be the main reason for the writing of this book. The author stresses very strongly the value of the "galactosurie provoquée" test in all the above types of jaundice and points out that it is important to consider the percentage of galactose in the 24 hour specimen of urine rather than the actual quantity of galactose excreted in the urine. The conclusions at the end of the book are concerned mainly with this test. It is recommended as the method of choice for following liver function during the course of any jaundice and is of chief value in associated severe hepatitis.

The third part of the book consists of a few commentaries on liver insufficiency in jaundice and the rôle the liver plays in the production of jaundice.

The fourth part consists of thirty-one case reports of various types of jaundice in which the different functional tests have been done. The "galactosurie provoquée" test is stressed again in this section.

This book is an interesting treatise on some of the tests available in the study of liver function in jaundice but too much is claimed for the galactosuria test the author describes. This, because he bases his conclusions on only thirty-one cases, in the majority of which only one or two determinations have been done. Further data should be gathered and, until then, this should be considered as a preliminary report.

Skin Diseases and Nutrition (Including the Dermatoses of Children). By ERICH URBACH, M.D. Authorized English Translation by Frederick Rehm Schmidt, M.D. (55 Illustrations, 8 diagrams, and 10 tables.) Wilhelm Maudrich, Publisher, Vienna, 1932. Price $7.00, postage free.

No other book covers the relationship of skin disease to nutrition in a comparable manner. The first half covers the general aspects of the relationship, with considerable emphasis on the chemical factors, reviewing dermatoses due to malnutrition,

due to dysfunction or disease of the digestive tract, metabolic disturbances, nutritive idiosyncrasies, etc. The author has done much chemical work in this field and has reviewed thoroughly and cites abstracts of the literature on these various subjects.

The second half of the book takes up individual diseases and discusses the dietetic therapy of each in detail. There is much of interest regarding the physiological chemistry of the skin, the deficiency diseases and allergic conditions especially. The author contributes to the treatment of this latter group propeptan therapy in which propeptans or specific peptones are utilized for desensitization. Infantile eczema of various types and the relation to feeding are discussed in some detail. The Gerson diet and its modifications in tuberculosis are commended. Numerous plates and tables add to the value of the book. The abstracts of other work are a distinct feature.

A Handbook of Experimental Pathology. By GEORGE WAGONER, M.D., and R. PHILIP CUSTER, M.D. Published by Charles C. Thomas. Springfield, Illinois. 1932. Pp. 160. Price $4.00.

This is a pioneer volume in its field, presenting experimental methods of illustrating certain of the more important problems in pathology. The problems are grouped into six sets of experiments in general pathology and ten sets of experiments in special pathology. While planned for the use of second year medical students, it is difficult to see how, if they were to follow this outline, they were to have time for any other work. It may be regarded as perhaps the dream of the experimental pathologist rather than of practical utility in undergraduate instruction.

For graduate students in the special field of pathology such a laboratory guide would be of great advantage. It is concise, clear and well planned. The author is to be congratulated on making available the outline of a definitely valuable course. Were all our medical students to become pathologists, this course might well form an important part of the curriculum. Under ordinary circumstances it is difficult to see how the work could be applied to undergraduate instruction, other than through aiding in the selection of a few important demonstrations for class purposes.

Diseases of the Nose, Throat, and Ear. Edited by A. Logan Turner with the collaboration of others. Third Edition, revised and enlarged. William Wood & Co., New York, 1932. Price $6.00.

This new edition, sponsored by a group of otolaryngologists in the Edinburgh Medical School, will be received with particular enthusiasm by general practitioners and medical students. The subject matter appears in a most legible type, with titles and subtitles which clearly mark it off with appropriate sections. Particularly outstanding are the illustrations, of which there are eight colored, full-page plates, reproducing most faithfully the diseases represented. The remaining smaller black and white drawings and photographs are all clear and instructive.

In a textbook of this size, some curtailment is necessary in respect to many of the more detached subjects, but this new edition nevertheless gives an entirely rewritten section on peroral endoscopy and on the physiology of the inner ear. It is pleasing to find the book well up-to-date in its consideration of the microscopic pathology of diseases of the ear, nose, and throat, there being some excellent reproductions of photomicrographs illustrating labyrinthine disease.

The appendix contains a large number of useful formulae and the index is pleasingly comprehensive. The book should serve a most useful function as a working guide for students, internes and practitioners who do not require a highly specialized and detailed reference book, but who will find of daily use a text covering common conditions and advocating well-recognized therapeutic measures in a lucid and handy form.

The Sex Technique in Marriage. By ISABEL EMSLIE HUTTON, M.D. Published by Emerson Books, Inc., 126 Maiden Lane, New York, N. Y. 160 pages. Price $2.00.

Among the books on sex hygiene that the reviewer has read, Dr. Hutton's little book impresses him as being the most practical.

The subject is handled very well; the writer makes her points clearly and without evasions or sentimentality. There is an introductory section on the anatomy of the sexual organs, followed by advice concerning the consummation of marriage and the sexual relations of later married life. Dr. Hutton takes up briefly several allied subjects, such as menstruation and the menopause, sterility and birth control. Her chapter dealing with this last topic is very sketchy, although her conclusions are sound. To the medically trained reader "The Sex Technique in Marriage" is likely to seem very elementary and perhaps incomplete, but to the prospective bride and groom, who are somewhat in the dark on these subjects, the book should be very helpful. To such, it may be recommended without hesitation.

Studies from the Rockefeller Institute for Medical Research. Reprints. Volume 83. New York. 1932. Pp. 571.

Of particular interest in this volume is the excellent summary of the filterable viruses by Rivers, which appeared in the July, 1932 number of *Physiological Reviews.*

A widely varying range of material is presented from the different departments of the hospital. In this particular group of reprints there is but little of immediate clinical interest.